Diseases of The Eye

1920

CONTENTS

SECTION I

ANATOMY AND PHYSIOLOGY

SECTION II

THE EXAMINATION OF THE EYE

SECTION III

DISEASES OF THE EYE

SECTION IV

ERRORS OF REFRACTION AND ANOMALIES OF ACCOMMODATION

SECTION V

DISORDERS OF MOTILITY OF THE EYE

SECTION VI

SYMPTOMATIC DISEASES OF THE EYE

SECTION VII

DISEASES OF THE ADNEXA OF THE EYE

SECTION VIII

PREVENTIVE OPHTHALMOLOGY

APPENDICES

DISEASES OF THE EYE

SECTION I

ANATOMY AND PHYSIOLOGY

CHAPTER I

Anatomy

THE sensory nerves of the body are provided with end organs, by means of which they receive specific physical stimuli and transform them into nerve impulses. The nerves of the special senses are no exceptions to the rule, and the eye is the highly differentiated and complex end organ of the sense of sight.

The wall of the globe is composed of a dense, elastic supporting membrane (Fig. 1). The anterior part of the membrane is transparent—the cornea; the remainder is opaque—the sclerotic. The anterior part of the sclerotic is covered by mucous membrane—the conjunctiva—which is reflected from its surface on to the lids.

The *cornea* consists of three layers: the epithelium, the substantia propria, and Descemet's membrane. The epithelium, which is stratified, may be regarded as the continuation of the conjunctiva over the cornea proper. It lies upon a homogeneous lamina of the substantia propria, called Bowman's membrane. The substantia propria may be regarded as the continuation forwards of the sclerotic. Descemet's membrane is a thin elastic membrane, covered on its posterior surface by endothelium: it may be regarded as the continuation forwards of the uveal tract. We shall see that the relationship of the three layers is of some pathological import-

ance, for when, as is often the case, the cornea suffers secondarily to some conjunctival complaint the epithelium and superficial layers are most likely to be affected : similarly, in diseases of the sclerotic the substantia propria suffers most, and in diseases of the uveal tract the endothelium, Descemet's membrane, and the adjoining posterior layers of the substantia propria.

The cornea is set into the sclerotic like a watch glass, *i.e.*,

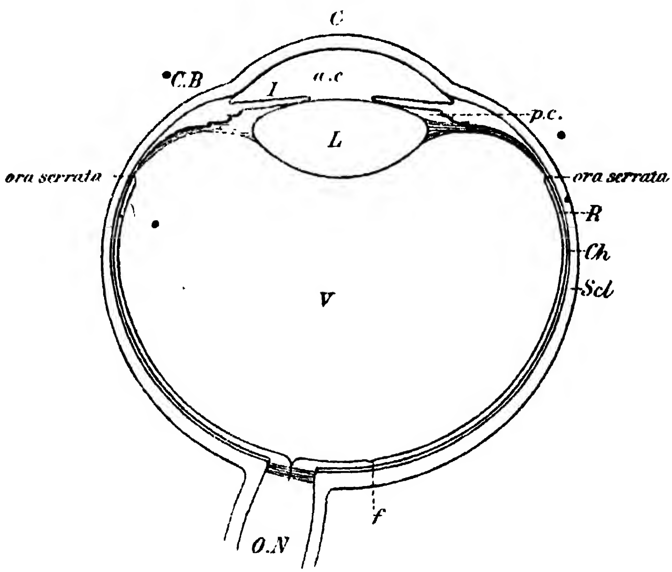


FIG. 1.—Diagrammatic horizontal section of the eye. C, cornea; *a.c.*, anterior chamber ; I, iris ; C.B., ciliary body ; *p.c.*, posterior chamber ; L, lens ; V, vitreous ; R, retina ; Ch., choroid ; Scl., sclerotic ; *f*, fovea centralis ; O.N., optic nerve.

the sclerotic overlaps the cornea all round the periphery. The cornea is very richly supplied with nerve fibres derived from the trigeminal. It has no blood vessels with the exception of minute festoons, about 1 mm. broad, at the periphery ; the cornea is therefore dependent for its nourishment upon diffusion of lymph, which is supplied from the conjunctival vessels.

Lining the sclerotic are two membranes : an outer, highly vascular, concerned chiefly in the nutrition of the eye, and comprising the greater part of the uveal tract ; and an inner,

nervous, the true visual nerve ending, concerned in the reception and transformation of light stimuli, and called the retina.

The uveal tract consists of three parts, of which the two posterior, the choroid and ciliary body, line the sclerotic while the anterior forms a free circular diaphragm, the iris. The plane of the iris is approximately coronal: the aperture

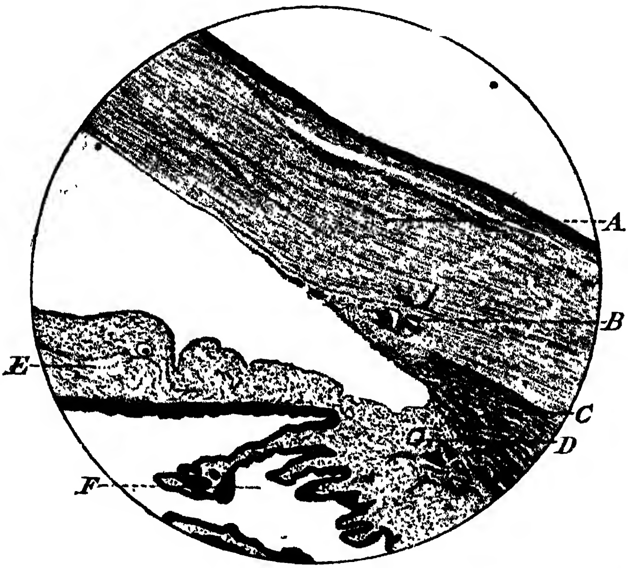


FIG. 2.—Angle of the anterior chamber. A, cornea; B, canal of Schlemm, which appears as several small spaces in the sclerotic just outside the ligamentum pectinatum iridis; it thus consists of irregular anastomosing venous channels which are cut across in the section; C, ciliary muscle; D, circulus arteriosus iridis major; E, iris; F, ciliary processes.

of the diaphragm is the pupil. Situated behind the iris and in contact with the pupillary margin is the crystalline lens.

The *anterior chamber* is a space filled with lymph, the *aqueous humour*; it is bounded in front by the cornea, behind by the iris and the part of the anterior surface of the lens which is exposed in the pupil. The sclerotic enters into the boundaries of the anterior chamber at the part which is known as the angle of the anterior chamber (Fig. 2). In the inner layers of the sclerotic at this part there is a network

of venous spaces which is called the *canal of Schlemm.* At the periphery, just anterior to the canal of Schlemm, Descemet's membrane splits up into fibrillæ, which are continuous with a meshwork of fibres stretching between the sclerotic and the iris, and known as the *ligamentum pectinatum iridis.* These fibres are covered by endothelium, which is continuous with that lining the cornea and also with that covering the iris. The spaces in the network of the ligamentum pectinatum iridis are called the *spaces of Fontana:* they are much better developed in lower mammals than in man. The tissue separating the ligamentum pectinatum from the canal of Schlemm is somewhat denser, and there is no free communication between the anterior chamber and the venous plexus, a thin membrane, covered on each surface by endothelium, being interposed. We shall see that a thorough knowledge of the anatomy of the angle of the anterior chamber is essential to the proper understanding of several pathological problems, especially that of glaucoma.

The anterior chamber is about 2·5 mm. deep in the centre in the normal adult: it is shallower in very young children and also in old people.

The *uveal tract,* as already mentioned, consists of the iris, the ciliary body, and the choroid, from before backwards.

The *iris* is composed of a stroma, consisting of branched connective tissue cells, usually pigmented, but unpigmented in blue irides, and containing a rich supply of blood vessels which run in a radial direction. The stroma is covered on its posterior surface by two layers of pigmented epithelium, which properly belong to the retina and are therefore called the *pars iridica retinæ,* or *pars retinalis iridis.* The anterior layer consists of flattened cells, which are very firmly attached to the stroma, the posterior of cubical cells, not so firmly attached to the anterior layer. Near the pupillary margin and concentric with it is a bundle of unstriped muscle fibres, the *sphincter iridis.* Associated with the anterior pigment epithelial cells there are fibres, arranged radially, which act as a *dilatator*

The anterior surface of the iris is covered with a single layer of endothelium, except at some minute depressions or crypts which are found most at the ciliary border. Here the lymph spaces between the stroma cells communicate directly with the anterior chamber: this is probably a device for ensuring rapid transference of lymph from the iris to the anterior chamber and *vice versâ,* so as to facilitate quick movements of

the pupil in response to variations in the intensity of the light falling upon the eye. The iris is thinnest at its attachment to the ciliary body, so that if torn it tends to give way here.

The iris is richly supplied by sensory nerve fibres derived from the trigeminal, a fact which it is important to remember, since touching or cutting the iris, especially if it is inflamed, is intensely painful. The sphincter iridis is supplied by motor nerve fibres derived from the oculomotor nerve, whilst the motor fibres of the dilatator iridis are derived from the cervical sympathetic nerve.

The *ciliary body* in antero-posterior section is shaped roughly like an isosceles triangle, with the base forwards. The iris

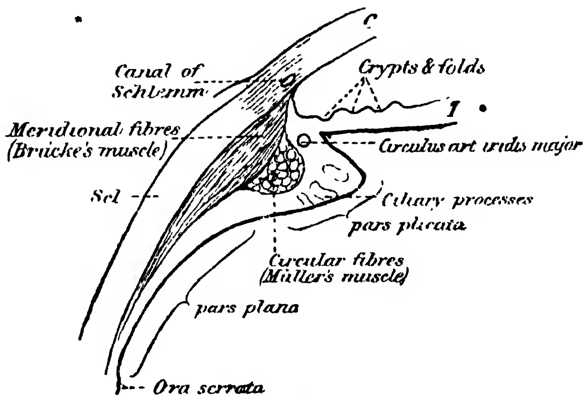


FIG. 3.—Diagrammatic meridional section of the ciliary body. Note that the sclerotic overlaps the cornea, as shown by the shading.

is attached to about the middle of the base, so that a small portion of the ciliary body enters into the posterior boundary of the anterior chamber at the angle (Fig. 3). The chief mass of the ciliary body is composed of unstriped muscle fibres, the *ciliary muscle.* This consists of two parts, an outer, in contact with the sclerotic, consisting of antero-posterior or meridional fibres, and an inner, consisting of fibres running at right angles to the former, arranged in a circle in the anterior part concentric with the base of the iris. The meridional fibres can be traced far back, well into the choroid, and are inserted anteriorly into a " spur " of the sclerotic, traction upon which by the muscle is thought to open up the canal of Schlemm.

If an eye is cut in half in an antero-posterior direction and the inner surface of the ciliary body is inspected, it will be

noticed that the anterior part has a number of folds upon it, while the posterior part is smooth. The anterior part is, therefore, called the pars plicata, the posterior, the pars plana. If the plications are counted with the naked eye or under slight magnification, it will be found that there are about seventy in the whole circumference. If microscopical sections are examined, innumerable smaller plications and processes, the *ciliary processes*, will be seen upon the pars plicata. These contain no part of the ciliary muscle, but consist essentially of tufts of blood vessels, not unlike the glomeruli of the kidney. They are covered upon the inner surface by two layers of epithelium, which belong properly to the retina, and are hence called the *pars ciliaris retinæ*. As in the pars iridica retinæ, the outer layer, corresponding with the anterior in the iris, consists of flattened cells, the inner of cubical cells, but unlike what obtains in the iris, they are not both pigmented, but only the outer layer.

The ciliary body extends backwards as far as the *ora serrata*, at which point the retina proper begins abruptly; the transition from ciliary body to choroid, on the other hand, is gradual, though this line is conveniently accepted as the limit of the two structures. The ora serrata is slightly more anterior on the nasal than on the temporal side.

The ciliary body is richly supplied with sensory nerve fibres derived from the trigeminal, so that great pain results from injury or acute inflammation. The ciliary muscle is supplied with motor fibres from the oculomotor nerve.

The *choroid* is an extremely vascular membrane in contact everywhere with the sclerotic, though not firmly adherent to it, so that there is a potential space between the two structures, which acts as a lymph space (Fig. 4). On the inner side, the choroid is covered by a thin elastic membrane, the *lamina vitrea*, or *membrane of Bruch*. The blood vessels of the choroid increase in size from within outwards, so that immediately beneath the membrane of Bruch there is a capillary plexus, the *choriocapillaris*. Following upon this is the layer of medium-sized vessels, while most external are the large vessels. The vessels are held together by a stroma consisting of branched pigmented connective tissue cells. It is easy to remember that the capillaries are innermost, because one of the chief functions of the choroid is to nourish the outer layers of the retina.

The choroid is supplied with sensory nerve fibres from the trigeminal.

The *retina* proper corresponds in extent with the choroid, which it lines. As already mentioned, however, and as shown by embryological research, it is continued forwards as a double layer of epithelium as far as the edge of the pupil. If the two layers of epithelium are traced backwards, the anterior layer in the iris is found to be continuous with the outer layer in the ciliary body, and this again is continued into the hexagonal pigment epithelium, which covers the membrane of Bruch. Similarly, the posterior layer in the iris, although pigmented, passes into the inner unpigmented layer of the ciliary body, and this suddenly changes at the ora serrata into the highly complex retina proper.

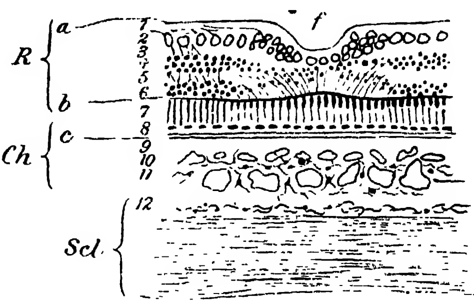


FIG. 4.—Diagrammatic section of retina, choroid, and sclerotic at posterior pole of the eye. *R*, retina, with *f*, fovea centralis: *a*, internal limiting membrane; 1, nerve fibre layer, 2, ganglion cell layer; 3, internal reticular layer; 4, internal nuclear layer; 5, external reticular layer; 6, external nuclear layer: *b*, external limiting membrane; 7, rods and cones; 8, retinal pigment epithelium. Ch., choroid: *c*, membrana vitrea or membrane of Bruch: 9, choriocapillaris; 10, layer of medium-sized vessels or Sattler's layer; 11, layer of large vessels or Haller's layer. Scl., sclerotic, with 12, lamina fusca on its inner surface.

The retina proper consists of a number of layers. Most external, in contact with the pigment epithelium, is a neural epithelium, the rods and cones (Fig. 4). Following this, in order from without inwards, are the outer nuclear, the outer reticular, the inner nuclear, the inner reticular, the ganglion cell, and the nerve fibre layers. These special nervous constituents are bound together by neuroglia, the better developed vertical strands being called the fibres of Müller. The interlacement of neuroglial fibrils on the outer side forms a sort of membrane which acts as a basement membrane for the rods and cones, the outer limiting membrane. Similarly on the inner side the bases of Müller's fibres spread out and form an inner limiting membrane upon the inner surface of the nerve fibre layer. Here the retina is in contact with the vitreous, which is generally said to have an extremely delicate

bounding membrane, the hyaloid membrane: this is probably only the denser outer layer of the vitreous gel, often modified by reagents.

At the optic disc the fibres of the nerve fibre layer pass into the optic nerve, the other layers of the retina stopping short abruptly at the edge of the porus opticus.

At the posterior pole of the eye, which is situated about 3 mm. to the temporal side of the optic disc, a specially differentiated spot is found in the retina of higher mammals (man and monkeys), the *fovea centralis*. As its name implies, it is a depression or pit, and here only cones are present in the neuro-epithelial layer and the other layers are almost completely absent. The fovea is the most sensitive part of the retina, and it is surrounded by a small area, the macula lutea, or yellow spot, which, though not so sensitive, is more so than other parts of the retina. It is here that the nuclear layers become gradually thinned out, while on the other hand parts of the reticular layers are specially in evidence: the ganglion cells too, instead of consisting of a single row of cells, are heaped up into several layers. There are no blood vessels in the retina at the macula, so that its nourishment here is entirely dependent upon the choroid (Fig. 5): by way of compensation the meshes of the capillary network of the choriocapillaris are particularly small here.

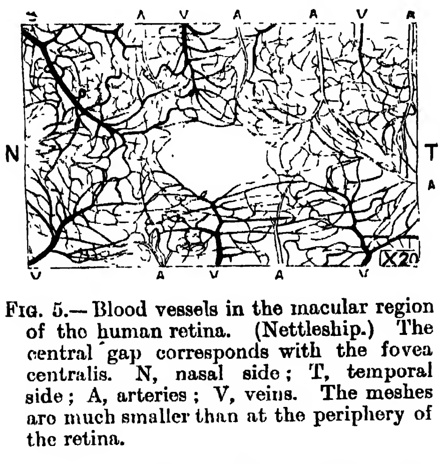


FIG. 5.—Blood vessels in the macular region of the human retina. (Nettleship.) The central gap corresponds with the fovea centralis. N, nasal side; T, temporal side; A, arteries; V, veins. The meshes are much smaller than at the periphery of the retina.

The so-called *optic nerve* is really a lobe of the brain, and therefore belongs properly to the central nervous system. Embryological and morphological investigations show that the bipolar cells of the retina, the nuclei of which are in the inner nuclear layer, probably correspond with the cells in the dorsal root ganglion of an ordinary sensory nerve (neurones of the first order). Similarly, the ganglion cells correspond morphologically with the cells of the nucleus gracilis, or nucleus cuneatus (neurones of the second order). Hence the

part of the visual nervous mechanism which corresponds with an ordinary peripheral sensory nerve is a microscopic cell with its processes situated within the retina itself. All the remainder is really part of the central nervous system, and we shall see that it responds to pathological processes more like the central than the peripheral nervous system.

The porus opticus is the aperture in the sclerotic through which the optic nerve passes. It varies much in shape in different cases, but in all it is traversed by a transverse network of connective tissue fibres containing much elastic tissue, the *lamina cribrosa.* The fibres of the nerve fibre layer of the retina pass through the meshes of the lamina cribrosa, and on the posterior side they suddenly become surrounded by medullary sheaths. These nerve fibres, which comprise the greater number of the nerve fibres in the so-called optic nerve, are the axis cylinder processes of the ganglion cells of the retina. They are therefore afferent or centripetal fibres, but the optic nerve also contains a few efferent or centrifugal fibres.

The *lens* is a biconvex mass of peculiarly differentiated epithelium. It will be remembered that it is developed from an invagination of the epidermal epiblast of the fœtus, so that what was originally the surface of the epithelium comes to lie in the centre of the lens, the peripheral cells corresponding with the basal cells of the epidermis. Just as the epidermis grows by the proliferation of the basal cells, the old superficial cells being cast off, so the lens grows by the proliferation of the peripheral cells. The old cells, however, cannot be cast off, but undergo change (sclerosis) analogous to that in the stratum granulosum of the epidermis, and become massed together in the centre or nucleus : moreover the newly formed cells elongate into fibres, the lens fibres, which have a rather complicated arrangement. Without going into details, it is important to bear in mind that the nucleus of the lens consists of the oldest cells and the periphery or cortex of the youngest. Further, it must be pointed out that at an early stage the productive basal cells become limited to a single row of cubical cells covering the anterior surface. The mass of epithelium which constitutes the lens is surrounded by a hyaline membrane, the lens capsule, which is thicker over the anterior than over the posterior surface (*v.* p. 50 ; Fig. 48) ; it is a cuticular deposit secreted by the epithelial cells.

The lens in fœtal life is almost spherical ; it gradually becomes flattened so as to assume the biconvex shape. It is

held in place by the suspensory ligament or zonule of Zinn. This is not a complete membrane, but consists of bundles of fibrils which pass from the surface of the ciliary body to the capsule. The flattening of the lens is due to these fibrils becoming more and more stretched as the eye grows. The fibrils pass in various directions and the various bundles often cross one another. Thus the most posterior arise from the pars plana of the ciliary body almost as far back as the ora serrata; these lie in contact for a considerable distance with the ciliary body and then curve towards the equator of the lens to be inserted into the capsule; most are inserted slightly anterior to the equator. A second group of bundles springs from the summits and sides of the ciliary processes, *i.e.*, far forwards, and passes backwards to be inserted into the lens capsule, slightly posterior to the equator. A third group passes from the summits of the processes almost directly inwards to be inserted at the equator.

It will be noticed that there is a somewhat triangular space between the back of the iris and the anterior surface of the lens, having its apex at the point where the pupillary margin comes in contact with the lens; it is bounded on the outer side by the ciliary body. This is the *posterior chamber*: it contains lymph of the same nature as the aqueous.

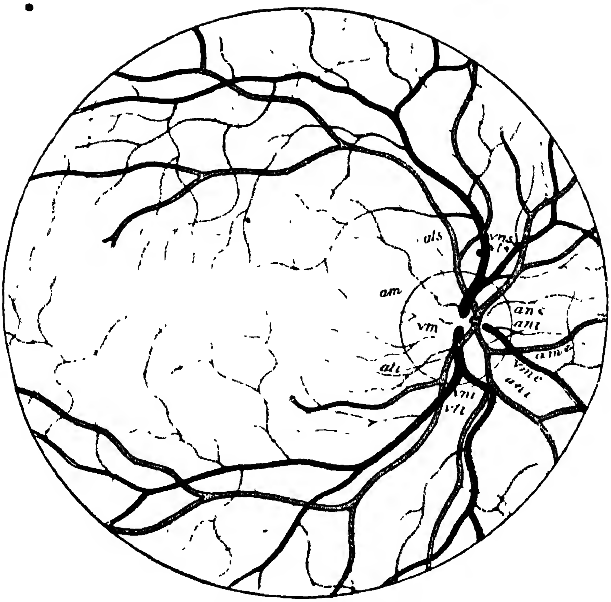
Behind the lens is the large vitreous chamber, containing the *vitreous humour*. This is a jelly-like material—in fact, it is probably an inert structureless gel—containing a few cells and wandering leucocytes. The fibres seen in histological sections are probably artefacts except in pathological conditions. As in other gels the concentration of the micellæ on the surface gives rise to the appearance of a boundary membrane in sections—the so-called hyaloid membrane.

The Blood Supply of the Eye

... ...ngement of the blood vessels which supply the eye is peculiar, and is of great importance in considering pathological conditions.

The arteries of the eye in man are all derived from the ophthalmic artery, which is a branch of the internal carotid. The ophthalmic artery has very few and insignificant anastomoses, so that on the arterial side the ocular circulation is an offshoot of the intracranial circulation. This is not the case to so marked a degree of the venous outflow of the eye. While in man most of the blood passes to the cavernous sinus by

PLATE I.

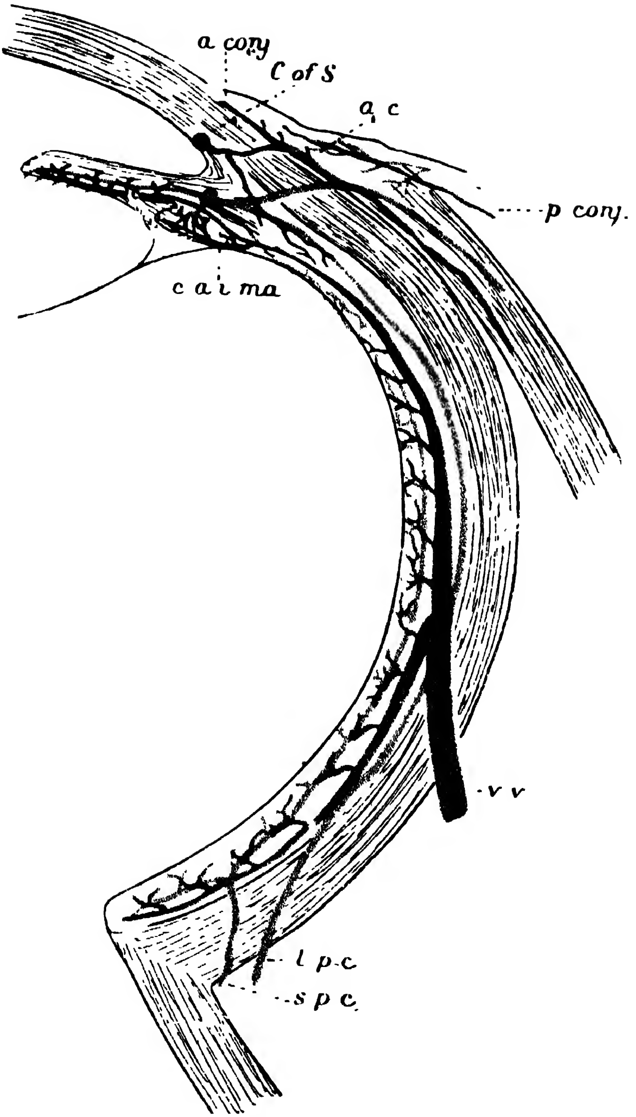


THE RETINAL VESSELS (after Leber).

a.t.s., superior temporal artery. *a.n.s.*, superior nasal artery. *a.t.i.*, inferior temporal artery. *a.n.i.*, inferior nasal artery. *a.m.*, macular artery. *v.t.s.*, superior temporal vein. *v.n.s.*, superior nasal vein. *v.t.i.*, inferior temporal vein. *v.n.i.*, inferior nasal vein. *v.m.*, macular vein.

[*To face p.* 10.

PLATE II.



THE CILIARY SYSTEM OF VESSELS (after Leber).

s.p.c., short posterior ciliary arteries. *l p.c.*, long posterior ciliary artery. *a.c.*, anterior ciliary vessels. *C. of S.*, canal of Schlemm. *c.a.i.ma.*, circulus arteriosus iridis major. *v.v.*, venae vorticosae. *a.conj.*, anterior conjunctival vessels. *p.conj.*, posterior conjunctival vessels.

way of the ophthalmic veins, yet it must be remembered that these anastomose freely in the orbit, the superior ophthalmic vein communicating with the angular vein at the root of the nose, and the inferior ophthalmic vein with the pterygoid plexus. Hence too great stress must not be laid upon the circulation in the retina as a guide to the condition of the intracranial circulation, as has been done in the past.

The retina is supplied by the central artery, which enters the nerve on its lower surface, 15—20 mm. behind the globe. The central artery divides on or slightly posterior to the surface of the disc into the main retinal trunks, which will be considered in detail later (Plate I.). The retinal arteries are end-arteries and have no anastomoses at the ora serrata. The only place where the retinal system anastomoses with any other is in the neighbourhood of the lamina cribrosa. The veins of the retina do not accurately follow the course of the arteries, but they behave similarly at the disc, uniting on or slightly posterior to the surface of the disc to form the central vein of the retina, which accurately follows the course of the corresponding artery.

The uveal tract is supplied by the ciliary arteries, which are divided into three groups—the short posterior, the long posterior, and the anterior (Plate II.; Fig. 6). The short posterior ciliary arteries, about twenty in number, pierce the sclerotic in a ring around the optic nerve, running perpendicularly through the sclera, to which fine branches are given off. The long posterior ciliary arteries, two in number, pierce the sclerotic slightly farther away from the nerve, in the horizontal meridian, one on the nasal, the other on the temporal side. They traverse the sclerotic very obliquely, running in it for a distance of 4 mm. The anterior ciliary arteries are derived from the muscular branches of the ophthalmic artery to the four recti. They pierce the sclerotic 5 or 6 mm. behind the limbus or corneo-scleral margin, giving off twigs to this region, to the conjunctiva and sclerotic.

The ciliary veins also form three groups—the short posterior ciliary, the venæ vorticosæ, and the anterior ciliary. The short posterior ciliary veins are unimportant; they do not receive any blood from the choroid, but only from the sclerotic. The venæ vorticosæ are the most important, consisting usually of four large trunks which open into the ophthalmic vein. They enter the sclerotic rather behind the equator of the globe, two above and two below: they pass very obliquely through the sclera. The anterior ciliary veins are smaller than the

corresponding arteries, since they receive blood only from the outer part of the ciliary muscle.

Of these ciliary vessels the short posterior ciliary arteries supply the whole of the choroid, being reinforced anteriorly

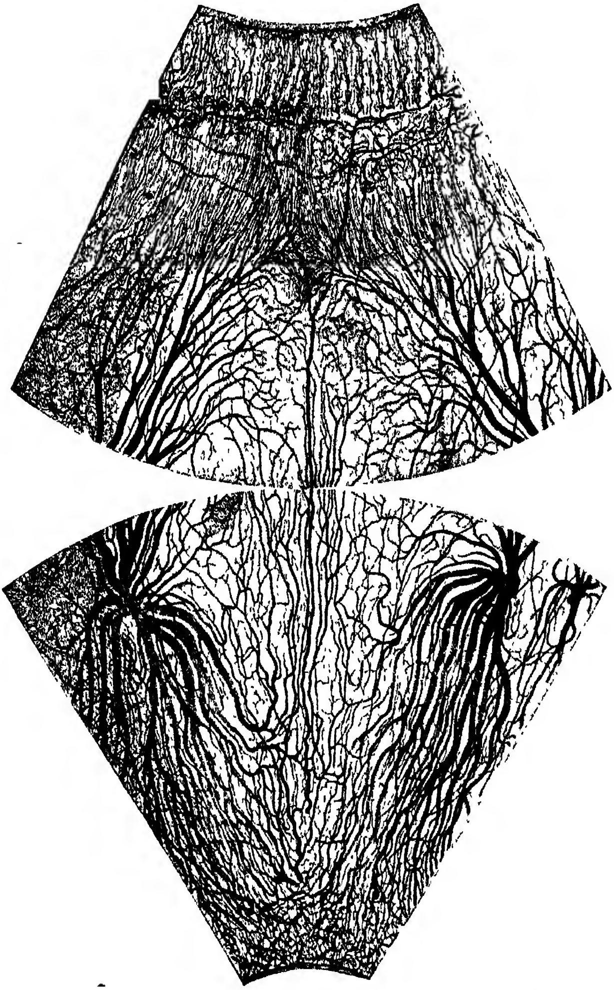


FIG. 6.—Blood vessels of the human uveal tract, injected, from the optic disc to the edge of the iris. (After Leber.) Arteries, black; veins, paler. Running up the centre is a long posterior ciliary artery. Two vorticose veins and their tributaries are seen. The capillaries are only partially filled in.

by anastomosis with recurrent branches from the ciliary body. The ciliary body and iris are supplied by the long posterior and anterior ciliary arteries. The blood from the whole of the uveal tract, with the exception of the ciliary muscle, normally leaves the eye by the venæ vorticosæ only.

The two long posterior ciliary arteries pass forwards between the choroid and the sclerotic, without dividing, as far as the posterior part of the ciliary body. Here each divides into two branches (Fig. 6): they run forwards in the ciliary muscle, and at its anterior part bend round in a circular direction, anastomosing with each other and thus forming the *circulus arteriosus iridis major*. This is situated in the ciliary body at the base of the iris: from it the ciliary processes and iris are supplied. Other branches from the major arterial circle run radially through the iris, dividing dendritically and ending in loops at the pupillary margin. A circular anastomosis takes place a little outside the pupillary margin, the *circulus arteriosus iridis minor*.

The tributaries of the vorticose veins, which receive the whole of the blood from the choroid, are arranged radially, the radii being bent, so as to give a whorled appearance—hence their name. The veins of the iris are collected into radial bundles which pass backwards through the ciliary body, receiving tributaries from the ciliary processes. Thus reinforced, they form an immense number of veins running backwards parallel to each other through the smooth part of the ciliary body. After reaching the choroid they converge to form the large anterior tributaries of the vorticose veins. The veins from the outer part of the ciliary muscle on the other hand pass forwards and unite with others to form a plexus, part of which is the so-called canal of Schlemm. These vessels drain into the anterior ciliary veins. The marginal loops of the cornea and the conjunctival vessels are branches of the anterior ciliary (Plate II.).

CHAPTER II

Physiology

In order that the eye may satisfactorily perform its duties as an organ of vision it is essential that a sharp image of objects in the outer world shall be formed upon the retina. This is effected by means of a series of curved surfaces, and the curvature of these surfaces and their relative positions to each other must be kept constant. For this purpose it is necessary that the walls of the globe should be kept stretched.

If a small canula connected with a narrow-bored mercury manometer is pushed into the anterior chamber or into the vitreous of an animal it will be found that the mercury in the manometer will rise about 25 mm. (Fig. 7). In other words, the contents of the eyeball, which are for the most part fluid, exert a pressure upon the inner side of the walls which is about 25 mm. of mercury greater than the atmospheric pressure which falls upon the outside of the walls; the walls are thus kept well stretched.

This pressure inside the eye is called the intraocular pressure, or the *tension*, of the eye. (These terms are used indiscriminately, though it is not quite accurate to do so.) It is obvious that it must be the fluid constituent of the contents of the globe which keeps up the internal pressure. This fluid fills the anterior and posterior chambers and permeates the vitreous. It is comparable to the lymph which bathes the tissues in other parts of the body, and it is indeed the lymph of the eye. In other parts of the body the chief function of the lymph is to carry food material to the tissue cells, and to carry away the effete products of the cell metabolism. It has a further function of keeping up the normal tissue tension. Both of these functions attain an unusual degree of importance in the case of the eye. We have already seen the necessity for keeping up the normal tension of the eye. As regards the nourishment of the cells, our review of the anatomy of the eye has shown that there are large areas, notably the whole of the lens and the vitreous, which possess

no blood supply. They are dependent entirely for their nutrition upon the lymph.

If water were to be forced into an impermeable elastic bag, the internal pressure might be kept indefinitely above the pressure upon the outside of the bag. Such an arrangement for keeping up the intraocular pressure would be unsatisfactory owing to the function of the lymph in nutrition. The stagnant fluid would soon lose all its food material, which would be used up, and it would become loaded with the excreted products of the cells, which would have a very deleterious effect upon them. Hence it is essential that the lymph shall be constantly renewed. This occurs in the eye. The lymph is continually being renewed, but at the same time it is equally rapidly being removed, and thus the amount present is kept constant.

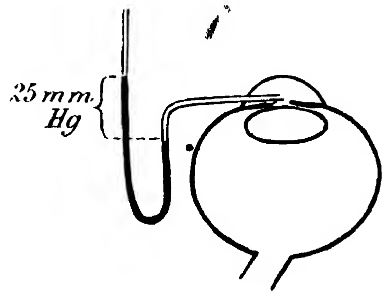


FIG. 7.—Diagram of manometer placed in communication with anterior chamber, showing that the normal intraocular pressure is about 25 mm. of mercury.

Of recent years the views of physiologists on the formation of lymph in the different tissues of the body have undergone great modification. It was natural that at first lymph formation should be regarded as a true secretion (Heidenhain). The typical example of secretion is the salivary gland. Here it is possible by stimulation of secretory nerves to obtain saliva at a pressure far exceeding that of the highest local intravascular pressure. The gland cells are doing work which cannot be accounted for by the ordinary physical laws of filtration and osmosis. Evidence of secretory nerves for lymph formation in the eye, as elsewhere, has proved negative. Moreover, it has been shown that the facts, both experimental and pathological, are not inconsistent with explanation on purely physical, " biophysical " and biochemical grounds.

Starling first produced definite evidence that lymph formation in the tissues could be explained by a simple process of filtration from the capillaries, the relations between the intracapillary and the lymph pressure, and the chemical constitutions of the lymph being in accord with this view. Leber arrived at the same conclusion with regard to the eye.

The advances of biophysics and biochemistry, however, have shown that this view must be modified. It has now been shown that many of the conditions of lymph production are consistent with the view that lymph is a simple dialysate from the blood plasma, and that the differences in constitution and pressure which obtain in different tissues may be due simply to the relative permeabilities of the dialysing membrane, *i.e.*, the capillary walls. These results, important as they are, do not of course solve the problem, for it still remains to account for the varying permeability of the capillary walls.

Moreover the composition and character of the intraocular fluid do not conform precisely with the requirements of a simple dialysate. It is to be remembered that in the formation of the aqueous, fluid has not only to pass through the capillary walls (the blood tissue barrier) but also the membranes and cellular layers separating the tissues essentially the uveal tract—from the chambers of the eye (the tissue-aqueous barrier). The existence of such membranes is proved, but about their properties or activities little is known, and whether these are governed by passive physico-chemical forces or are supplemented by active "secretory" processes has yet to be determined.

The fundamental fact is that the normal intraocular pressure, as shown by the manometer, is from 20 to 25 mm. Hg. above the atmospheric pressure. How is this brought about and maintained? The most obvious source of energy is the blood pressure derived from the heart beat. The first step is therefore to enquire into the hydrostatic conditions of the intraocular circulation. The blood in the human eye is derived entirely from the internal carotid artery. The pressure in this artery is very little less than that in the brachial artery—diastolic/systolic, 60–80/110–125 mm. Hg. The diastolic pressure in the ophthalmic artery can be measured in animals by increasing the intraocular pressure until the pulsation of the retinal arteries is maximal, the systolic by further increasing the pressure until the pulsation is abolished. It is found to be diastolic/systolic, 60–70/95–115 mm. Hg.

The pressure in the intraocular arteries has been measured by introducing a micropipette containing methylene blue into a retinal artery by the aid of a micro-manipulator, and ophthalmoscopic observation. The pipette is connected with a manometer, and fluid is forced in until it just appears in the vessel. Average pressures were: diastolic/systolic, 64/88 mm. Hg. (Duke-Elder).

Average results gave a mean fall of only about 10 mm. Hg. from carotid to ophthalmic artery, and of 25 mm. Hg. in the first branching in the eye, *i.e.*, 25 per cent. of the total pressure. The intraocular pressure shows a further fall of 54 mm. Hg.

In order that the circulation may be maintained it is clear that the lowest intraocular venous pressure must exceed the intraocular pressure. The difference, measured by the micropipette, is only about 2 mm. Hg. (Duke-Elder). A similar difference is found in Schlemm's canal.

The blood vessels of the eye are subject to variations in calibre as in other parts of the body. These are not merely passive as would be the case if the eyeball were a rigid closed box. There is definite proof that the intraocular arterioles are under the control of vaso-constrictor fibres derived from the cervical sympathetic, but there is no evidence of the existence of vaso-dilatator fibres. Vaso-motor changes, however, have been proved to be due to the liberation of substances which act upon the neuro-myal junction. In the case of sympathetic fibres the substance is adrenaline or a nearly allied substance; in that of the parasympathetic system acetylcholine or an allied substance. Vaso-dilatation, however, can be brought about indirectly by antidromic impulses in sensory nerves (Bayliss), and by axon reflexes. These lead to the liberation of histamine or an allied substance, which causes relaxation of the smaller vessels and capillaries.

There is no means of measuring the intracapillary pressure within the eye directly. The old view that variations in the calibre of the capillaries is passive owing to changes in the calibre of the arterioles has been profoundly modified by the researches of Krogh, Dale, Landis and others, who have shown that relatively enormous variations in capillary pressure occur, both rapidly and in neighbouring areas, and that these are brought about chiefly by dynamic changes in the size of the capillaries, probably effected by Rouget's cells and by local chemical changes due to acetylcholine or histamine. The local effect of histamine, liberated by irritation of the fifth nerve endings, and the more distant effect due to axon reflexes (which is abolished by cocaine) have been proved to occur in the iris by Duke-Elder. Great variations in intracapillary pressure between the limits of 70–80 mm. Hg. (arterial) and 20–25 mm. Hg. (venous) are possible, and it may be that in some circumstances it rises as high as 50 mm. Hg.

Vaso-dilatation produced by histamine cannot be counter-

acted by vaso-constrictors, such as adrenaline: moreover, it causes marked increase in the permeability of the capillary walls, as evidenced by wheals in the skin, etc. This increased

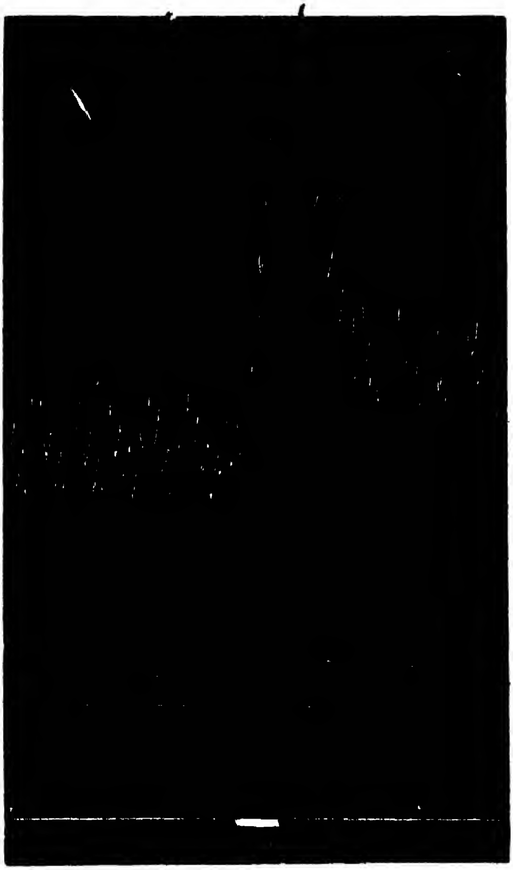


Fig. 8.—Tracing of right carotid blood pressure, intraocular pressure from canula in anterior chamber of left eye of a dog, which was fully under the influence of morphia, A.C.E. mixture, and curare. Stimulation of the vasomotor centre, showing that the intraocular pressure follows passively all the changes in the general blood pressure as produced by Traube-Hering curves and constriction of the arterioles of the splanchnic area.

permeability is of great pathological importance, accounting for plasmoid exudates in inflammatory conditions, etc.: it may be beneficial, opening the gate for the passage of immune

bodies, all of which are large-moleculed colloids, and some drugs (*e.g.*, arsenic compounds).

Manometric observations show that in general the intraocular pressure follows changes in the general blood pressure passively (Fig. 8). Thus the large rises of blood pressure produced by stimulation of the vaso-motor centre, splanchnic nerves, asphyxia, injection of nicotine or adrenaline, etc., are reflected in the intraocular pressure curve. The parallelism, however, is not absolute, as was shown long ago by the proof of the existence of vaso-constrictor nerves and the local effects of adrenaline. Perfusion experiments on the isolated head have revealed other local effects due to axon reflexes, chemical substances such as choline, histamine, drugs, etc. Probably most of these effects are due to changes in the capillary circulation.

The intraocular fluid differs from lymph in other tissues of the body in its remarkable poverty in proteins. In still greater degree does it differ thus from the blood plasma, but the differences are purely quantitative, all the constituents of the blood plasma being present in the aqueous. The proteins are very much reduced (0·2 per cent. as compared with 7·36 per cent. in serum), much more so than in any other tissue lymph ; but the relative proportions of globulin to albumin are the same as in the plasma. Non-dissociated diffusible substances, such as sugar, urea, etc., are equally partitioned between aqueous and plasma. Dissociated diffusible substances, *e.g.*, metallic salts, which are split up into electrically charged ions in solution are unequally distributed, the cations being in less and the anions in greater concentration in the aqueous. The greater concentration of chlorides has long been known and should have attracted greater attention by the advocates of the filtration theory, since it militates against that theory. It is however consistent with the dialysation theory, negatively charged ions being driven through the capillary membrane in order to maintain thermodynamic equilibrium with positively charged colloid ions (proteins) in the blood. For the same reason the aqueous is slightly more acid than the plasma (*p*H 7·2 : 7·3). The plasma exerts an osmotic pressure of 20–30 mm. Hg. greater than the aqueous owing to the excess of non-diffusible colloids in it.

What may be called the static intraocular pressure is therefore the resultant of the intracapillary blood pressure minus the difference between the osmotic pressures of aqueous and plasma (*e.g.*, 50 mm. Hg. − 30 mm. Hg. = 20 mm. Hg.). The intraocular pressure however is never static. It follows

passively normal changes in the blood pressure due to the pulse wave and respiration. It undergoes large rises due to the activity of the extrinsic ocular muscles and the movements of the lids, especially of the orbicularis palpebrarum. Large changes in the general blood pressure are accurately reflected in it except in so far as they are modified by local vasomotor or chemical effects. If, however, the general changes persist, as in cases of high blood pressure, compensation occurs and the intraocular pressure resumes its normal level. Convection currents are also set up in the anterior chamber owing to the difference in temperature between the iris and cornea, the aqueous moving upwards in front of the iris and downwards behind the cornea. These currents can be seen with the slit lamp (*vide* p. 97) when, as is often the case, the aqueous contains particles in suspension.

The atomic and molecular changes which have already been discussed are therefore associated with molar movements which alter the local hydrostatic conditions. These are specially marked under abnormal conditions. If the aqueous is evacuated, *e.g.*, by paracentesis (*vide* p. 209), the capillaries dilate, the walls become more permeable and filtration of fluid takes place. The fluid thus formed more nearly resembles the blood plasma in containing more protein than the normal aqueous. If again the eye is massaged the intraocular pressure falls. Seeing that this pressure is dependent upon the volume of the contents of the globe and the other conditions are not materially altered, the fall of pressure must be due to expulsion of fluid from the eye. There is therefore some mechanism for the filtration of fluid out of the eye. Cases of secondary glaucoma (*vide* p. 280) indicate the chief site of filtration out of the eye, for in them the angle of the anterior chamber is blocked. There can be no doubt that the rise in intraocular tension which occurs in secondary glaucoma is due to blockage of this filtration angle, whereby the aqueous is denied free access to the canal of Schlemm.

It has been shown that if the intraocular pressure is raised so as to exceed that of the intravenous pressure, the veins collapse. This results in a reflex rise of intravascular pressure so that the circulation is restored. This process may in some cases be repeated until the intraocular pressure equals the arterial pressure, when the circulation stops. The canal of Schlemm, however, is a venous channel in the substance of the cornea and therefore does not collapse even when the intraocular pressure is moderately raised. Filtration can therefore

occur through its inner wall—unless the latter is rendered impermeable by adherent iris, etc.; an exit is thus provided for the excessive fluid and the normal intraocular pressure is restored. The meridional fibres of the ciliary muscle (*vide* p. 5, Fig. 3) are inserted anteriorly into a scleral "spur," and there is reason to think that when this muscle contracts in accommodation it pulls this spur backwards, thus tending to keep the canal of Schlemm open.

The permeability of the capillaries is increased in inflammatory conditions, *e.g.*, iridocyclitis (*vide* p. 256), and a plasmoid lymph, rich in protein, is formed. This causes a rise in intraocular pressure, and there can be little doubt that since in these cases the filtration angle is not otherwise obstructed and is indeed generally abnormally open the high tension is due to the difficulty of filtration of the large molecular colloid proteins into the canal of Schlemm.

We may therefore conclude that both dialysation and filtration play their parts in the formation and disposal of the intraocular fluid and the maintenance of the intraocular pressure. They do not suffice to explain all the facts of normal and abnormal intraocular pressure. The dialysation theory alone has to fall back upon the variable permeability of the capillary walls in the various tissues of the body to explain the differences of lymph pressure and constitution; and this variable permeability is at present only explicable on teleological grounds. Moreover, it must be supplemented by a fuller knowledge of the membrane-system forming the barrier between the vascularized tissues and the chambers of the eye.

CHAPTER III

Elementary Optics

It has already been stated that sharp images of external objects must be formed upon the retina if the latter are to be seen clearly. Before considering how this is effected it will be advisable to refresh the reader's memory of the elementary principles of optics. I would seriously impress upon the student that success in the diagnosis, and hence in the treatment, of diseases of the eye is impossible if such elementary principles of optics as are set forth here are not thoroughly mastered.

If white light, such as sunlight, is passed through a suitable prism or diffraction grating a spectrum is formed, consisting of rays differing from each other in wave-length. Of these certain are visible and appear to the majority of people as pure colours, viz., red, orange, yellow, green, blue, and violet in the order named, the red having the longest and the violet the shortest wave-length. The visible spectrum extends from about 723 $\mu\mu$ at the red end to 397 $\mu\mu$ at the violet end, or roughly from 700 $\mu\mu$ to 400 $\mu\mu$. Beyond the red end are infra-red rays of greater length which, when absorbed, cause a rise in temperature and are commonly known as heat rays. Beyond the violet end are waves of smaller length, the ultra-violet rays, which are capable of causing chemical action. The longer visible rays also cause a rise in temperature, and the visible rays are also actinic, though less so than the infra-red and ultra-violet respectively. Glass absorbs some of the heat rays and many of the ultra-violet. Prisms and lenses made of quartz allow most of the ultra-violet rays to pass unimpeded. The media of the eye are uniformly permeable to the visible rays between 660 $\mu\mu$ and 390 $\mu\mu$. With regard to ultra-violet rays, the cornea absorbs all rays beyond 295 $\mu\mu$, the lens all rays beyond 350 $\mu\mu$, the vitreous shows an absorption band with its maximum at 270 $\mu\mu$ (E. K. Martin). Rays between 400 $\mu\mu$ and 295 $\mu\mu$ can therefore reach the lens, those between 400 $\mu\mu$ and 350 $\mu\mu$ can reach the retina in the normal eye, and those between 400 $\mu\mu$ and 295 $\mu\mu$ can reach the retina in the aphakic eye. Whenever absorption occurs there is the possibility of pathological changes resulting. Sunlight at the lower sea-levels is poor in ultra-violet rays, which fall off

rapidly in intensity beyond 380 $\mu\mu$. Ordinary glass used for spectacles absorbs rays beyond 350 $\mu\mu$. Heat radiation from 1,100 $\mu\mu$ to 700 $\mu\mu$ passes into the eye almost unchecked, and a large amount of it reaches the retina (Hartridge and Hill). The pigment epithelium on the back of the iris absorbs heat radiation of all wave-lengths, and the same is probable of the retinal epithelium at the back of the eye.

It is a familiar fact that a candle flame emits light in all directions. The light is transmitted in straight lines, so that we may imagine the light coming from the candle as consisting of an immense number of straight lines, all intersecting in some part of the flame. If we consider a minute point in the flame, then all the straight lines which cross in this point may be said to diverge from it. Each of these hypothetical straight lines is called a ray.

Now, every point on such a ray represents, or is the image of, the point of light from which it springs. This is shown very clearly by a simple experiment carried out in a dark room. Make a pinhole in a piece of cardboard (Fig. 9, A), and hold the cardboard in front of the candle (C) at a little distance from it. Beyond the cardboard hold up a white screen (B), so that the cardboard is between the screen and the candle. A dim image (D) of the flame will be thrown upon the screen, and it will be noticed that it is upside down, *i.e.*, an inverted image of the flame is formed. This is due to the fact that the cardboard cuts off all the rays of light from the candle except such as can pass through the hole. The only rays from the top of the flame which can pass through the hole are those which are caught upon the lower part of the screen. They represent the top of the flame; hence they reproduce its shape accurately. The image is very dim because only a few rays of light can pass through the small hole. Now make another hole a little distance away from the first. Another inverted image of the flame is seen. If a dozen holes are made, a dozen images appear. If two holes are very close together the images will overlap. If a large hole is made, so many images overlap that all resemblance to the original flame is lost, and part of the screen becomes uniformly illu-

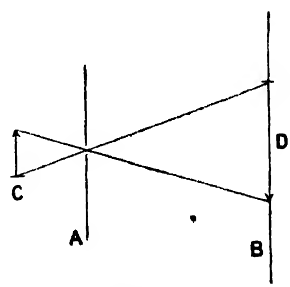


FIG. 9.

minated. If we take away the cardboard altogether the whole screen becomes illuminated, and we now know that this is because we have an infinite number of images of the flame all overlapping each other.

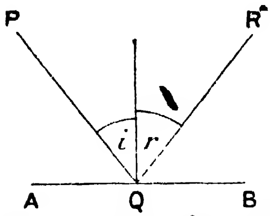


FIG. 10.—The ray from P which strikes the mirror AB at Q is reflected to R, so that PQ and QR are in the same plane, viz., that of the paper, and the angle of incidence, *i*, is equal to the angle of reflection, *r*.

Light travels with different velocities in different media. If the velocity is less in one medium than another the first medium is said to be optically denser than the second.

When light, travelling in one medium, meets another medium it breaks up into two parts: part is *reflected* back into the first medium; part is *refracted* into the second medium. If the second medium is opaque none of the light is refracted.

REFLECTION

Let us now consider what happens to a ray of light when, travelling in one medium, it is reflected from the surface of a denser medium. We have already said that its direction is altered. Before it meets the surface it is called an incident ray; after it leaves the surface it is called the reflected ray. If a line is drawn at right angles to the surface at the point where the incident ray meets it, it is found to be an invariable rule that the incident ray makes the same angle with this line, which is called the normal, that the reflected ray makes with it. Put in formal language, this law of reflection is that *for all surfaces the angle of incidence is equal to the angle of reflection, and is in the same plane with it* (Fig. 10).

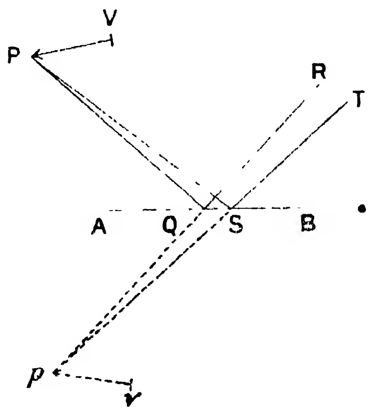


FIG. 11.

Plane Mirrors. Let us apply this rule to an ordinary flat mirror (Fig. 11). If P is a luminous point in front of the mirror AB, the ray PQ will be reflected towards R, and the ray PS towards T; *i.e.*, the reflected rays QR and ST appear to come

from p, a point as far behind the mirror as P is in front of it. As the rays QR and ST have to be produced backwards in order that they may meet, no real image is formed, and such an image is called a virtual image. Note that the rays reflected from a plane mirror are divergent. The same reasoning holds good for every point on the object PV, its image being pv as far behind the mirror as the object is in front of it: moreover, the size of the image is equal to that of the object.

Concave Mirrors. Here the normal to the surface is the radius of the sphere. If AH (Fig. 12) is part of the section of a concave mirror and PB is an object, K being the centre of the sphere, then the line HKB is called the axis, and H the apex of the mirror. The ray PK through the centre of the sphere will obviously be reflected along itself, so that the image of P must be on PK. The ray PA, parallel to the axis, will meet PK in p. Hence p is the image of P. Now it is found

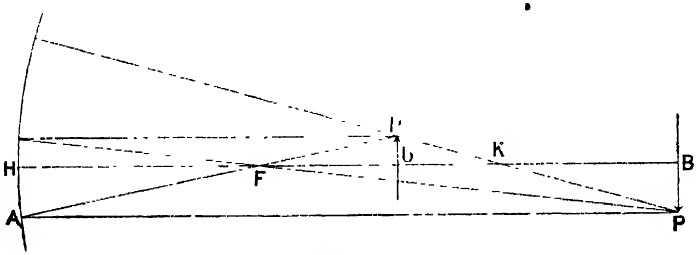


Fig. 12.

that all rays parallel to the axis and not very far removed from it cut the axis in the same point, F, and this point bisects the line HK. This point is called the principal focus of the mirror. If the object PB were removed a very great distance away from the mirror, all the rays which fell upon a small portion of the mirror near H would diverge so little from each other that they would all be practically parallel to BH, and the image of PB would be extremely small and situated at F. In each of these cases the image is an inverted one of the object.

It is an axiom of optics that the direction of the rays is reversible. Hence, if pb were an object, it would have its image at PB, and if there were an object at F, all the rays from it reflected by the mirror would be parallel to the axis, and the image would be infinitely large and situated at infinity.

What would happen if the object were situated between F and H? In that case (Fig. 13) the rays would diverge after reflection as if they came from an object behind the mirror,

much as they do with a plane mirror. The image would therefore be a virtual one, situated behind the mirror: it would be erect and larger than the object.

The important fact to remember with regard to concave mirrors is that if the object is farther away from the mirror than its focal distance, *i.e.*, than half its radius of curvature,

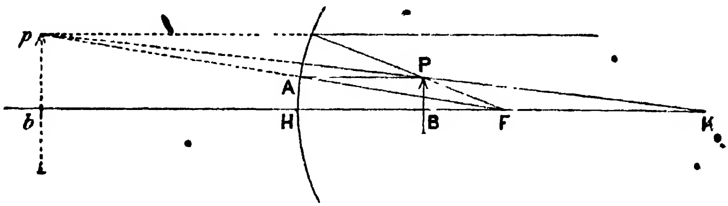


FIG. 13.—The ray from P parallel to the axis is reflected through F, the principal focus. The ray FP is reflected parallel to the axis. The ray KP is normal to the surface, and is therefore reflected on itself. Any two of these rays will give the situation of *p*, the image of P.

the image is a real inverted one situated also in front of the mirror. This is the condition which is almost always present in the ordinary use of ophthalmic instruments.

Convex Mirrors. We are not accustomed to use convex mirrors in ophthalmic instruments, but it is necessary to know what happens with them, since the cornea acts as a convex

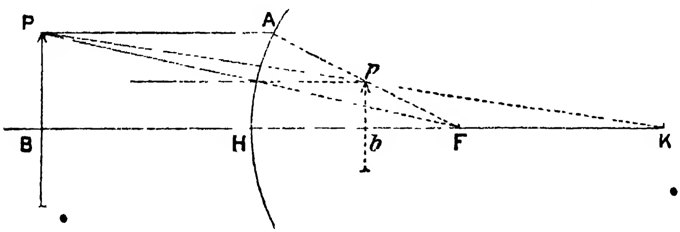


FIG. 14.—Reflection by a convex mirror. The description of Fig. 13 applies equally to this case.

mirror. Here, as will be seen from Fig. 14, the image is always virtual, erect, and smaller than the object. As with the concave mirror, if the object is a long way off, the image will be situated at the principal focus, *i.e.*, at a distance equal to half the radius of curvature behind the mirror.

REFRACTION

We have now to consider what happens to the refracted ray when the incident ray, travelling in one medium, *e.g.*, air,

meets an optically denser medium, *e.g.*, glass. We have already said that the light will now travel more slowly. It follows directly from this fact that it will be deviated towards the normal to the surface, and it will be more deviated the greater the difference in optical density between the two media. If the density of air is taken as unity, then the ratio of its density to that of the second medium is called the *index of refraction* of the medium.

Plane Lamina. Let us see what happens when an incident ray, such as PQ (Fig. 15), meets the surface of a plate of glass with parallel sides. It will be deflected towards the normal, *ab*. When the ray passes out of the glass on the other side, it will obviously be deflected away from the new normal, *cd*, just as much as PQ was deflected towards it. Hence the emergent ray RS will be parallel to the incident ray PQ. If the plate of glass is very thin, RS will be practically continuous with PQ.

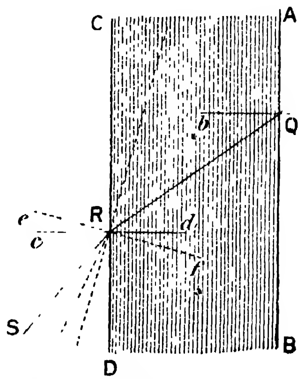


FIG. 15.—Refraction by a plane lamina.

Prisms. If we imagine one side (CD) of the plate in the last figure to revolve round R, we shall be able to understand the nature of refraction by a prism. QR will now make a larger angle with the new normal *ef* than it did with the old one *cd*. Consequently the angle of refraction will also be larger, *i.e.*, the new direction of the emergent ray will be RT. In other words, the ray is deviated towards the base of the prism.

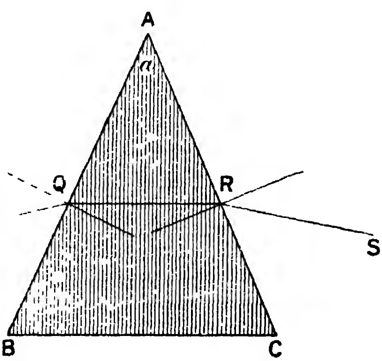


FIG. 16.—Refraction by a prism.

The ray PQRS in Fig. 16 is said to pass symmetrically

through the prism. In these circumstances, if the prism is made of crown glass, the deviation of the ray is approximately equal to half the refracting angle of the prism, *a*.

Prisms are numbered according to the angle of the prism ($^\circ$), or preferably according to the actual deviation ($^\Delta$), *e.g.*, a 4° prism is approximately the same as a 2^Δ prism.

We are accustomed to project objects along the direction of the rays of light as they enter the eye, and in doing so we ignore the effect of refraction, since it enters relatively little into our everyday experience. If, therefore, we look at a candle P through a prism, as in Fig. 17, the light will appear to come from *p*. Objects, then, seen through a prism, appear displaced towards the apex of the prism.

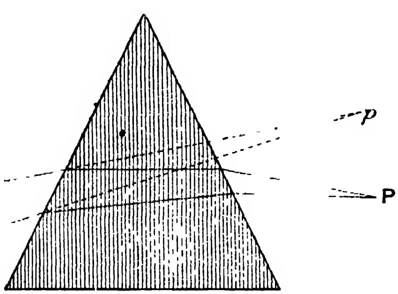


FIG. 17.—Displacement of objects seen through a prism. The object P appears to be situated at *p*.

Lenses. Ordinary lenses are pieces of glass with spherical surfaces. The line passing through the centres of curvature of the surfaces is called the axis of the lens. Fig. 18 shows the chief varieties of lenses, viz., (1) biconvex, (2) biconcave, (3) plano-convex, (4) plano-concave, (5) convexo-concave or meniscus: these names require no further explanation.

The effect of a biconvex lens upon rays of light meeting it

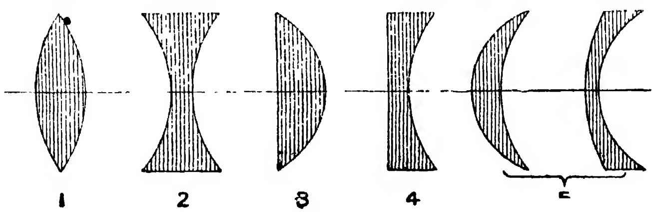


FIG. 18.—Types of lenses.

is very similar to what would occur if it were replaced by two prisms set base to base (Fig. 19).

If the incident rays are parallel to the axis they will be

refracted in such a manner that they all cross the axis in a single point upon the other side of the lens. This point is called the principal focus of the lens, and its distance from the lens is called the focal distance or length of the lens. When the lens has the same medium, *e.g.*, air, on each side of it, the

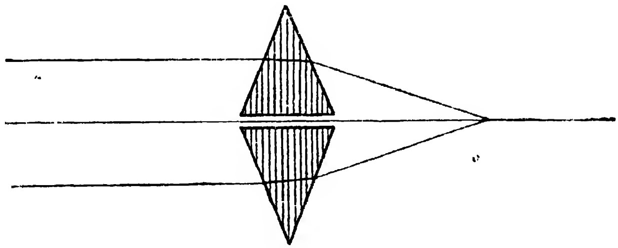


FIG 19.

two principal foci, one on each side of the lens, are situated at equal distances from it. For thin glass lenses of low power the focal distance is equal to the radius of curvature of the two surfaces when these are equally curved. If there is an object a very long distance away from the lens, the rays which come from it are practically parallel. Hence in this case an image of the object will be formed by the lens at its principal focus;

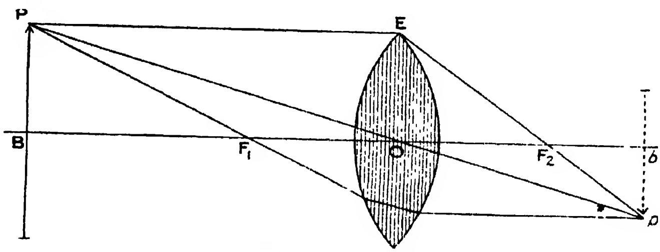


FIG. 20.—The ray PE, parallel to the axis, is refracted through the second principal focus F_2. The ray PF_1, through the first principal focus, is refracted parallel to the axis. The ray PO, through the optical centre of the lens, is not deflected. Any two of these rays give the situation of *p*, the image of P.

it will be inverted and very small. If the object is gradually brought nearer and nearer to the lens (Fig. 20) the image will recede farther and farther from it; from being very small it will grow larger and larger, until, when the object is at the principal focus, the image will have receded to infinity, and it will be infinitely large, *i.e.*, all the rays coming from the object

at the principal focus are parallel to the axis and to each other after refraction. If the object is brought still closer to the lens than its focal distance (Fig. 21) it will be found that its image is a virtual one behind the object, and that it is erect and larger than the object. The positions of the object and image bear a constant relationship to each other and are called conjugate foci.

There is a point in the middle of a biconvex lens which is

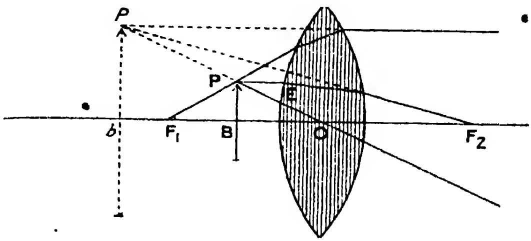


FIG. 21.—The description of Fig. 20 applies equally to this figure.

called its optical centre. With thin lenses any ray which passes through this point is practically not deviated at all. It is easy to understand why this is so. If PQRS (Fig. 22) is such a ray and tangents are drawn to the two surfaces at the points Q and R, these two tangents will be parallel to each other. Consequently, the lens acts for such a ray exactly as if it were a plate with parallel sides, and we have already seen that in such a case the emergent ray is parallel to its original direction. If the lens is very thin the refracted ray will be practically continuous with the incident ray.

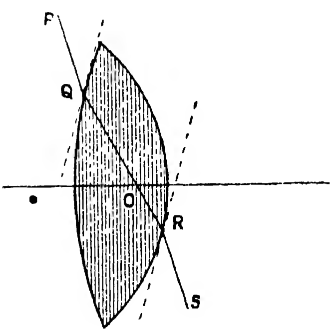


FIG. 22.—Properties of the optical centre of a lens.

If we know these facts, viz., that rays passing through the optical centre are not deviated, and that rays passing through the principal focus are parallel to the axis after refraction, we can easily construct the image of an object in any given position. Thus, in Fig. 20, if PB is an object, the ray PO through the optical centre O will not

be deviated; the ray PE parallel to the axis will pass through the second principal focus F_2; and the ray PF_1 through the first principal focus will be parallel to the axis after refraction. Hence *pb* must be the image of PB.

The effect of a biconcave lens upon rays of light meeting it

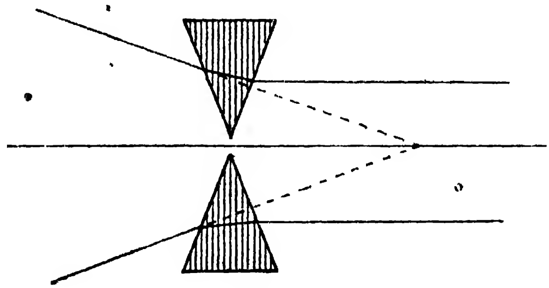


FIG. 23.

is very similar to what would occur if it were replaced by two prisms set apex to apex (Fig. 23).

Here, if the incident rays are parallel to the axis they will be divergent after refraction, and the amount of divergence of the individual rays will be such that if they are produced backwards they will all cross the axis in a single point upon the same side of the lens that they came from. This and the

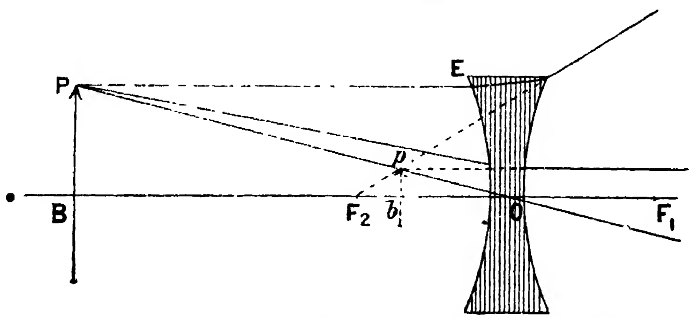


FIG. 24.—The description of Fig. 20 applies equally to this figure.

corresponding point on the other side of the lens are called the principal foci. The biconcave lens also has an optical centre, situated upon the axis within it and having the same properties as in the case of the convex lens. The image of any object formed by a concave lens can be constructed in exactly the same manner as for a convex lens (Fig. 24). It will be found that in every position of the object the

image is always virtual, erect and smaller than the object.

Plano-convex and plano-concave lenses act like biconvex and biconcave respectively, but in them the optical centre is on the curved surface at the point where the axis cuts it. Menisci act as convex or concave lenses according as the convex or the concave surface has the greater curvature. In them the optical centre is outside the lens.

It will have been noticed that the refractive power of a lens varies inversely as the focal distance, *i.e.*, a lens with a short focal distance will bend the rays more than one with a longer focal distance. It is necessary to have some system of numbering lenses so as to indicate their refractive power. The most convenient system for ophthalmic purposes is that which takes a lens with a focal distance of 1 metre as a standard. Such a lens is said to have a refractive power of 1 *dioptre.* A lens with a focal length of half a metre will be twice as strong as one whose focal length is 1 metre: the refractive power of such a lens is therefore 2 dioptres. Similarly, a 3 D (3 dioptre) lens has a focal length of one-third of a metre, or 33 cm.; a 4 D lens, 25 cm.; and so on. It is important to remember that in this system the standard is a metre, not a centimetre or a millimetre; otherwise confusion may arise.

Lenses were formerly numbered according to their focal lengths measured in inches. Since the inch has a different value in different places, the method is unsatisfactory. Prescriptions for spectacles are, however, sometimes met with in this notation. They are easily transformed into the dioptric system by remembering that there are 40 inches (roughly, or 36 Paris inches) in 1 metre. Therefore a 40 inch lens = 1 D; a 20 inch lens = 2 D; a 4 inch lens $= \frac{40}{4} = 10$ D; and so on: a lens of focal length = 4 Paris inches $= \frac{36}{4} = 9$ D.

Convex lenses are indicated by a plus sign (+), concave by a minus sign (−) before the number.

Cylindrical lenses are also used in ophthalmology; their nature and use will be considered at a later stage.

We often wish to find out whether a lens is convex or concave, and what its refractive power is. There are several ways of doing this, but the simplest is with the assistance of the trial case. Hold a convex lens up near the eye and look at distant objects through it; then move the lens a little from side to side: the distant objects will seem to move in the

opposite direction to that in which the lens is moved. If we repeat the process with a concave lens the objects seem to move in the same direction as the lens. The reason is to be found in the fact that a convex lens forms an inverted, whilst a concave forms an erect, image. If we place two lenses of opposite sign but equal curvature in contact with one another the combination will make a plate with parallel sides: such a plate, as we know, does not practically deflect the rays of light at all. Hence we can determine the strength of a lens by exactly neutralising it with a lens of the opposite sign out of the trial case. Let us take a concrete example, a particular lens which we wish to determine. We hold it up and find that distant objects seem to move in the opposite direction to the lens. We know that it is a convex lens. We then put a weak concave lens in contact with it and repeat the process. We find that with a $-$ 2 D lens objects still seem to move in the opposite direction, though not so much. With a $-$ 3 D lens there is only a trace of movement, and with a $-$ 3·5 D lens there is no movement at all. We conclude that the original lens was + 3·5 D. In performing this test it is important to have the two lenses as closely in contact as possible, and also

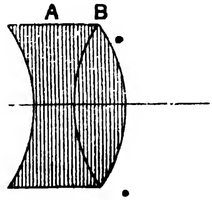


FIG. 25.

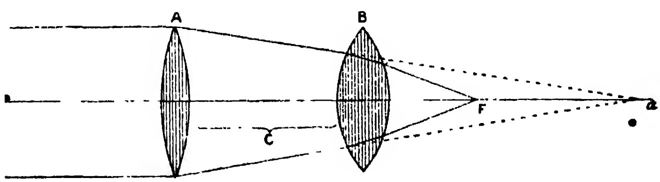


FIG. 26.

to have their centres in contact (Fig. 25). If the centre of one lens is higher than that of the other they will obviously not counteract each other exactly. If they are not in contact the result will be either too high or too low.

When the lenses are in contact the refractive power of the combination (D) is equal to the algebraical sum of the refractive powers of the two lenses (d_1, d_2): *i.e.*, $D = d_1 + d_2$, or $\frac{1}{F} = \frac{1}{f_1} + \frac{1}{f_2}$ where F, f_1, f_2, are the respective focal distances (Fig. 26).

Suppose, however, that two convex lenses are separated by a distance c (Fig. 26). The lens A will make parallel rays converge towards a, but after a distance c they meet the lens B: hence the convergence of the rays is not expressed by $\frac{1}{f_1}$, but by $\frac{1}{f_1 - c}$. Therefore the combined effect of the lenses, D, or $\frac{1}{F}$, is now equal to $\frac{1}{f_1 - c} + \frac{1}{f_2}$.

If the second lens (B) is a concave one (Fig. 27) its effect will

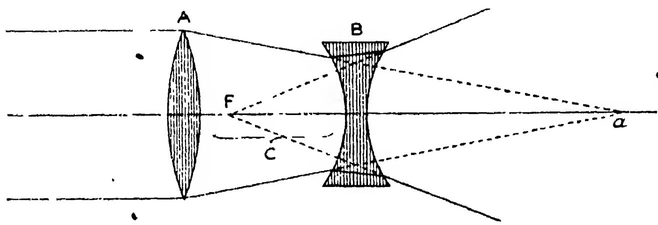


FIG. 27.

be one of divergence, so that it must have a negative sign, and D will now be equal to $\frac{1}{f_1 - c} - \frac{1}{f_2}$.

It is to be noted that in the formula

$$\frac{1}{F} = \frac{1}{f_1 - c} + \frac{1}{f_2}$$

F is now the posterior focal length, the incident light impinging upon the lens whose focal length is f_1 and being directed towards the lens whose focal length is f_2. The following formula gives the equivalent focal length (F_e) of the combination, irrespective of the direction of light:—

$$F_e = \frac{f_1 f_2}{f_1 + f_2 - c}$$

Examples: (1) $f_1 = 333$ mm.; $f_2 = 250$ mm.; $c = 133$ mm.

$$\text{Then } \frac{1}{F} = \frac{1}{333 - 133} + \frac{1}{250} = \frac{1}{111}$$

That is, the combination of a + 3 D lens with a + 4 D, separated by a distance of 133 mm., will be that of a + 9 D lens, instead of a + 7 D if they had been in contact.

(2) $f_1 = 333$ mm.; $f_2 = -83$ mm.; $c = 133$ mm.

$$\text{Then } \frac{1}{F} = \frac{1}{333 - 133} - \frac{1}{83} = -\frac{1}{142}$$

That is, the combination of a + 3 D lens with a — 12 D, separated by a distance of 133 mm., will be that of a — 7 D lens, instead of — 9 D if they had been in contact.

CHAPTER IV

Elementary Physiological Optics

The eye as an optical instrument very much resembles an ordinary photographic camera. The latter consists of a dark chamber with an aperture in front containing a strong convex lens, and with a movable back behind. The effect of the lens is exactly like that shown in Fig. 20. PB will represent the object to be photographed; the movable back is adjusted so that it occupies the position of *pb*, in which case a sharp image of the object will be thrown upon the ground glass which forms the back. The ground glass is then replaced by a sensitive plate, and the photograph is taken.

In the eye the retina corresponds with the sensitive plate. Instead of having only one lens in the front aperture, represented by the crystalline lens, there is also a curved plate with parallel sides, the cornea, which acts like another lens, and indeed has a much stronger optical effect than the crystalline lens. The object of this more complicated arrangement is to shorten the focal distance of the system, so that the eye may be shorter and more compact.

From this analogy we see that the eye, from the optical point of view, acts like a strong convex lens. We have already stated that when a lens has the same medium on each side of it the anterior and the posterior focal distances are equal to one another. This is not the case in the eye. Here the medium in front is air, while behind the lens there is the vitreous, which has a higher refractive index, rather more than that of water. Hence the anterior and posterior principal focal distances are not equal, the anterior being about 15 mm. in front of the cornea, and the posterior about 24 mm. behind it.

The cornea has about the same optical density or refractive index as the aqueous, which is also equal to that of the vitreous. The anterior surface of the cornea may be regarded as nearly spherical, the radius of curvature being 8 mm. The centres of curvature of the cornea and the two surfaces of the lens are all on the same straight line, which is called the *optic*

axis. When a ray of light meets the cornea (Fig. 29) the ray will be deflected towards the normal, *i.e.*, towards the radius drawn through the point of incidence. It will pass through the layers of the cornea in the new direction, and will also continue in the same direction through the aqueous, for, as we have said, the refractive index of the aqueous is the same as that of the cornea. When the ray meets the lens, which has a greater refractive index than the aqueous, it will again be deflected in the same sense, *i.e.*, towards the axis upon which the cornea and lens are centred.

The eye approximates to a homocentric optical system, *i.e.*, one composed of a series of spherical surfaces whose centres lie on a common *axis*. The geometrical properties of such a system may

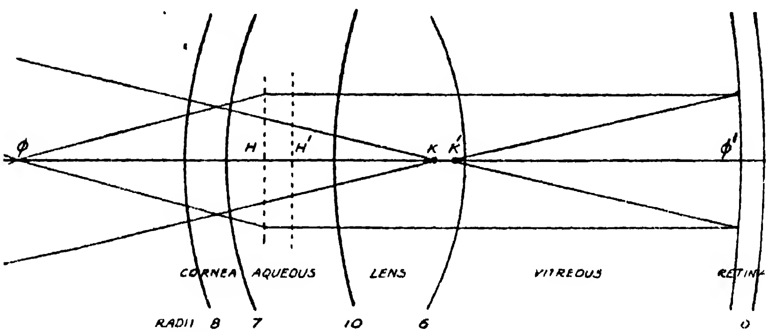


FIG. 28.—THE CARDINAL POINTS OF THE EYE.

φ, The anterior principal focus in front of the cornea.
φ', The posterior principal focus upon the retina.
H, H', the principal points, in the anterior chamber.
K, K', the nodal points, in the posterior part of the lens.
R.I. Refractive indices.

be much simplified by considering them to possess three pairs of *cardinal points* or *planes*. Rays passing through either *principal focus* emerge from the system, after refraction, parallel to each other. A ray directed through one *nodal point* emerges, after refraction, through the second nodal point and parallel to its original direction. A ray passing through any point on one *principal plane* emerges, after refraction, as though it came from the corresponding point on the second principal plane, but the incident and emergent rays are not usually parallel to each other.

Since the principal and nodal points in the eye are very close to each other no great error is introduced by fusing them into a

single principal point at the point where the axis cuts the cornea and a single nodal point in the posterior part of the lens (*e.g.*, Fig. 30).

We have seen that in the case of a lens, and the same is true of any homocentric optical system, parallel rays meet at

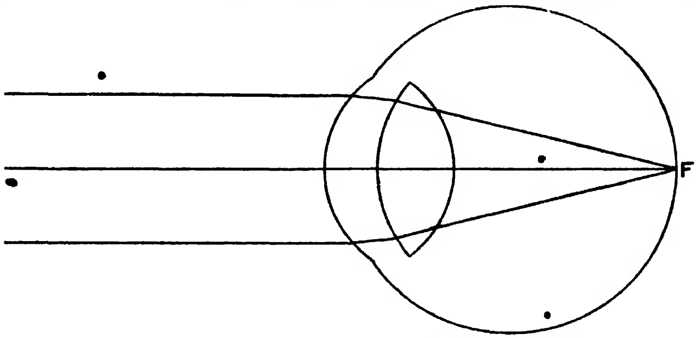


FIG. 29.—Emmetropic eye. Parallel rays are brought to a focus on the retina.

the principal focus. Hence, if parallel rays fall upon the cornea, they will be brought to a focus 24 mm. behind it. Now, the rays which are emitted by a luminous body are divergent. If, however, the object is a long distance away, the individual rays in any small bundle will diverge so little from each other that they may be regarded as practically

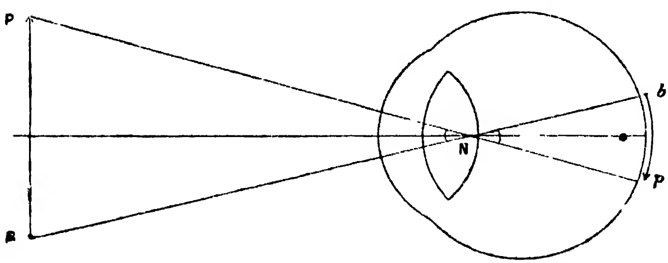


FIG 30.—Nodal point of the eye, visual angle, and relative sizes of object and retinal image.

parallel. This is the case with the small bundles of rays which are able to enter the pupil of the eye. Hence, as in the case of a convex lens (*vide* p. 29), the image formed by the eye of these distant objects will be situated at the principal focus, *i.e.*, 24 mm. behind the cornea. But that is exactly the distance of

the retina from the cornea in the normal eye. Hence we see that the normal eye in its condition of rest is so constituted that distant objects form their images upon the retina (Fig, 29).

The optic axis, produced backwards to meet the retina, cuts it almost exactly at the fovea centralis. Hence, any distant object on the prolongation forwards of the optic axis will have its image at the fovea, which is the best spot for distinct vision.

We notice that, just as with a convex lens, the image is inverted. It is re-inverted psychologically in the brain.

It is easy to find the size of the retinal image which any external object will form, since the eye possesses an optical centre, which, however, is usually called the *nodal point* (N), quite similar to the optical centre of the lens. In the eye this point is situated upon the optic axis near the back of the lens.

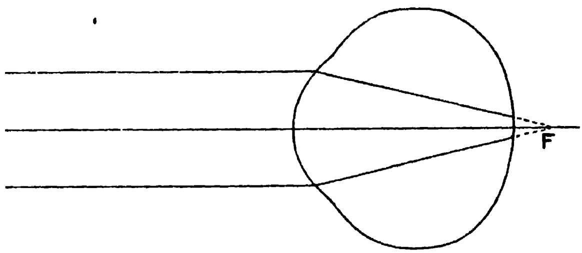


FIG. 31.—Hypermetropic eye. Parallel rays tend towards a focus behind the retina.

As in the case of lenses, any ray which passes through this point will not be appreciably deflected. If, therefore, there is an object PB (Fig. 30) in front of the eye, the size of its retinal image *pb* is found by joining the extremities of the object and the nodal point and producing these lines until they meet the retina. The lines will enclose an angle, PNB, which is called the *visual angle ;* in other words, the angle subtended by the object at the nodal point is called the visual angle. It is of course equal to the angle *p*N*b*, which is subtended by the retinal image at the nodal point.

In some eyes the retina is not situated in exactly the right place for the images of distant objects to be clearly focussed upon it. It may be too far forward (Fig. 31), or too far back (Fig. 32) ; in the former case they are said to be hypermetropic, in the latter myopic. If we consider the effect upon parallel rays we shall see that in the hypermetropic eye they have not had space to come to a focus, whereas in the myopic

eye they have not only come to a focus, but have commenced to diverge. In each case a blurred image will be formed upon the retina, and vision will be impaired. Such conditions are called errors of refraction or *ametropia* (ἀ, privative, μέτρον, measure ; not according to measure). In contradistinction to hypermetropia and myopia the normal condition is called emmetropia.

It has already been stated that in optics the direction of the rays is reversible. Let us imagine a minute point on the retina to be luminous. It will give out rays which will diverge in all directions. Some of these rays will meet the lens and cornea and pass out of the eye. Now, in the emmetropic eye, those rays which get through the pupil will have to submit to exactly the same optical deviations as the parallel rays falling upon the cornea did when they passed into the eye and came

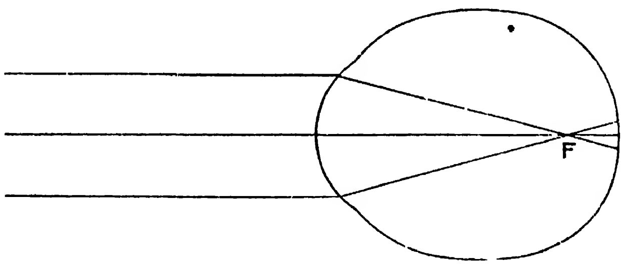


FIG 32.—Myopic eye. Parallel rays are brought to a focus in front of the retina.

to a focus on the retina. Hence, on the principle of the reversibility of the rays, the rays coming from a point on the retina will be parallel to each other when they leave the eye (Fig. 29).

Suppose, however, that the eye is hypermetropic owing to being too short (Fig. 33). The rays coming from a point on the retina will be relatively more divergent than the corresponding rays of the emmetropic eye before they fall upon the back of the lens. (Compare the effect of placing an object closer to a convex lens than its principal focus (Fig. 21).) The lens and aqueous and cornea will therefore cause them to converge less than in the emmetropic eye. They will therefore still be divergent when they leave the eye, though of course not so divergent as when they were passing through the vitreous. In fact, their direction will be the same as if they came from a point behind the eye. The nearer the

retina is to the lens, the more divergent they will be, and the nearer to the back of the eye will be the point from which they seem to come. This virtual point (R) behind the eye is called the *remote or far point* of the eye. The point on the retina and this point behind the eye are really conjugate foci (Fig. 33).

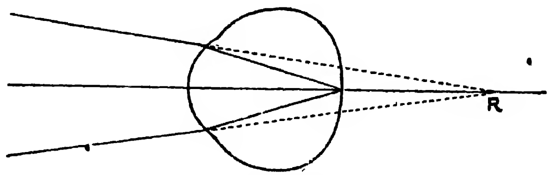


FIG. 33.—Hypermetropic eye. Rays from a point on the retina are divergent when they emerge from the eye, as if they came from the point, R, behind the eye.

Suppose now that the eye is myopic owing to being too long (Fig. 34). The rays coming from a point on the retina will be relatively less divergent than the corresponding rays of the emmetropic before they fall on the back of the lens. (Compare the effect of placing an object farther away from a convex lens than its principal focus (Fig. 20).) The refractive media in front will therefore cause them to converge more than in

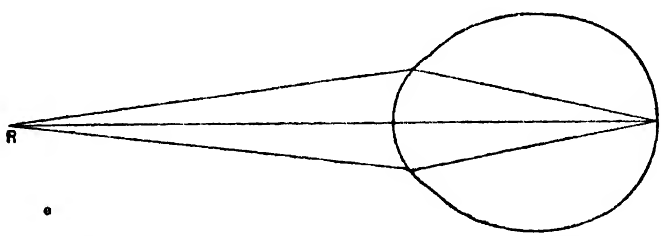


FIG. 34.—Myopic eye. Rays from a point on the retina are convergent when they emerge from the eye, so that they cross at a real point, R, in front of the eye.

the emmetropic eye. They will hence be convergent when they leave the eye, and will cross in a point (R) somewhere in front of the eye (Fig. 34). The farther the retina is from the lens, *i.e.*, the higher the degree of myopia, the more convergent they will be, and the nearer to the front of the eye will be the point where they cross. This point is again the conjugate focus to the point on the retina, but in this case it is a real point. It is also called the remote or far point of the eye.

Where then is the far point of the emmetropic eye? We have seen that in each of the other conditions it is where the rays emitted from a point on the retina meet after emerging from the eye. In the emmetropic eye the emergent rays are parallel to each other. But parallel rays meet at infinity; therefore the far point of the emmetropic eye is at infinity.

It is obvious that, in hypermetropia, if we give the rays the requisite amount of convergence before they enter the eye they will be brought to a focus upon the retina. We can do this by placing a convex lens in front of the eye (Fig. 35). This is what is done by means of spectacles. The refractive or convergent power of a convex lens is the reciprocal of its focal distance. Hence in hypermetropia of 1 D, a convex lens of 1 D or 1 metre focal distance placed in contact with the cornea will direct parallel rays towards a point 1 metre

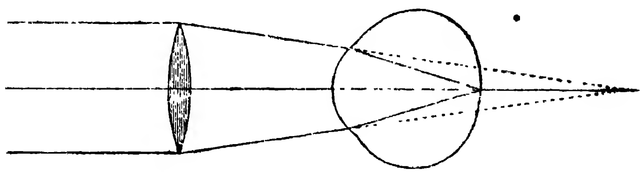


FIG. 35.—Hypermetropic eye. Parallel incident rays brought to a focus on the retina by means of a suitable convex lens.

behind the eye, *i.e.*, to the far point of the eye. Such a lens acting in combination with the refractive force of the eye would bring the rays to a focus on the retina. But lenses can only rarely be worn in contact with the cornea (*v.* p. 538). If the lens is placed 20 mm. in front of the cornea its focal length will have to be 1,020 mm. instead of 1,000 mm. (*vide* p. 34), but this small difference is negligible, and we are accustomed to measure errors of refraction by the strength of the lens which is required when it is placed in the ordinary position of a spectacle lens (Fig. 35).

Similarly in myopia, if we give the rays the requisite amount of divergence before they enter the eye they will be brought to a focus upon the retina. We do this by placing a concave lens in front of the eye (Fig. 36). Here we should want a – 1 D lens in contact with the cornea to correct a myopia of 1 D, *i.e.*, an eye whose far point is 1 metre in front of the eye. If the glass is worn about 20 mm. in front of the eye it will have to be rather stronger, *i.e.*, it will have to be of a focal distance of 980 mm. instead of 1,000 mm.

There is an advantage in having the correcting glass in axial ametropia in the position of the anterior focus of the eye, because under these conditions the size of the retinal image is exactly the same as if the eye were emmetropic (Figs. 37, 39). The anterior focus is about 15 mm. in front of the eye. The optician aims to place the optical centre of the spectacle lens 12–13 mm. from the cornea. We have already discovered (p. 41) that the farther the glass is from the eye the convex glass in hypermetropia has to be weaker, and the concave glass in myopia stronger. There is also an effect on the size of the retinal image. If the glass is more than 15 mm. from the cornea the retinal image in hypermetropia is larger, and in myopia smaller than the emmetropic image (Figs. 38, 40). The increase in size in hypermetropia is advantageous, but the diminution in myopia is a disadvantage, especially in very

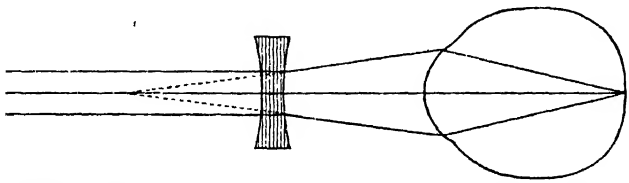


FIG. 36.—Myopic eye. Parallel incident rays brought to a focus on the retina by means of a suitable concave lens.

high degrees. Consequently in the latter the glasses ought to be made to fit as closely to the eyes as possible, the eyelashes being cut short if necessary.

We have seen that in every case the far point and a point on the retina are conjugate foci. Hence an object situated at the far point of any eye will have a sharp image upon the retina (Fig. 34). This may be made clearer perhaps if we consider the myopic eye from another point of view. We have seen that the rays from a point on the retina meet in front of the eye at the far point. We may again use the principle of reversibility of rays. If there is a luminous point at the far point, the rays emitted from it which enter the eye will meet on the retina; in other words, the image of an object at the far point will be upon the retina.

From these considerations we learn how it is that a patient with myopia cannot see clearly things which are a long distance away, whereas he can see things which are near. In common parlance, he is "short-sighted." He can see things at a distance better if he screws up his eyes. This is because

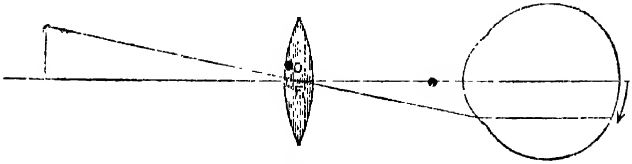


Fig. 37.

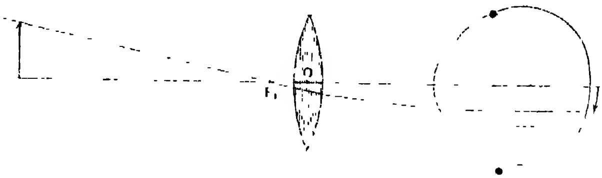


Fig. 38.

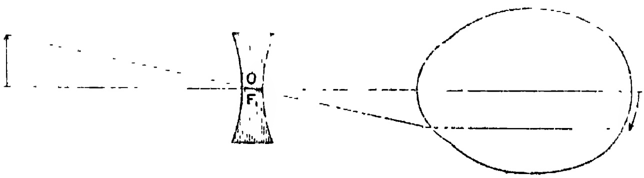


Fig. 39.

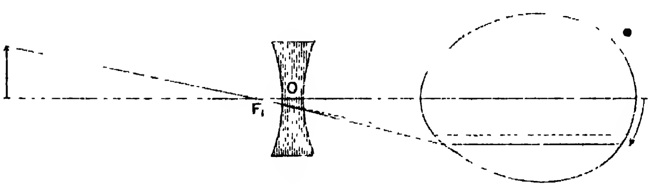


Fig. 40.

Figs. 37-40.—Effect of correcting lenses upon the size of the retinal image. In Figs. 37, 39, where the optical centre of the lens, O coincides with the anterior focal point of the eye, F_1, the size of the retinal image is the same as in emmetropia. When the lens is closer to the eye than the anterior focal distance of the eye the size of the retinal image is diminished (convex lens, Fig. 38) or increased (concave lens, Fig. 40).

he thus makes a narrow slit to look through, and this slit acts like the hole in the cardboard before a candle (*vide* p. 23). The term myopia originated in this peculiarity (μύειν, to shut; ὤψ, the eye or countenance).

The patient with hypermetropia, on the other hand, can see neither distant nor near objects clearly with his eyes at rest, since the far point is virtual, and it is impossible to place an object at its situation. We shall see later that he is better off than the myope by virtue of accommodation.

We have already seen that the emmetrope sees only distant objects clearly with his eyes at rest, since the rays from such distant objects are nearly parallel. For practical purposes

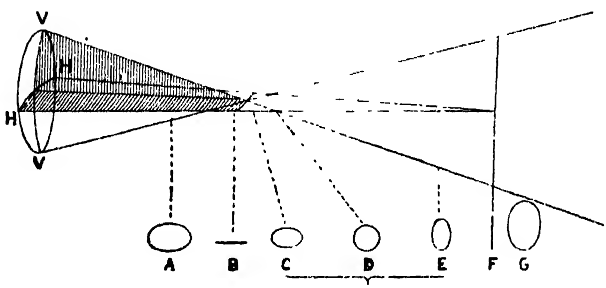


Fig. 41.—Sturm's conoid. V V, vertical meridian of refracting surface, more curved than H H, the horizontal meridian. A, B, C, D, E, F, G, sections of conoid. From B to F is the focal interval of Sturm. D shows the circle of least diffusion.

objects more than 6 metres (20 feet) away from the eye form clear images upon the retina.

The condition of an eye, whether emmetropic, hypermetropic, or myopic, is called its refraction, or more accurately its static refraction, since the term applies to the eye at rest.

We have hitherto considered only such errors of refraction as are due to axial shortening or lengthening of the eye (*axial ametropia*). It is not difficult to understand that ametropia might be due to other causes. Thus, myopia might be due to the refractive power of the eye being too strong; in this case parallel rays would be brought to a focus in front of the retina even if this were in its normal position. Increase or decrease in the refractive power of the eye might be due to two causes. It might be due to alteration in the refractive

indices of the media, or to alteration in the curvatures of the refractive surfaces: ametropia due to these causes is called *index* or *curvature ametropia* respectively. Both are much less common than axial ametropia. Index ametropia is very rare, though we shall have a physiological example of it later (*vide* p. 53).

Curvature ametropia has a special importance, not because it gives rise to simple hypermetropia or myopia, but because it is the cause of another very troublesome error of refraction, called *astigmatism*. In most eyes, even if they are ametropic, the areas of the refractive surfaces uncovered by the pupil and used in vision are very nearly spherical. Sometimes, however, they are not. In most of these cases it is the cornea which is at fault, and the error is generally of such a nature that this surface is flatter from side to side than it is from above downwards. Even in these cases the curvatures in the vertical and horizontal meridians are both spherical, but the radius of curvature of the horizontal meridian is longer than that of the vertical. Such a surface is said to have a *toric* curvature. Perhaps the pressure of the lids on the globe tends to squeeze it above and below.

What will be the effect of such a toric cornea upon the refraction of the eye? Clearly the more curved meridian will have more refractive or convergent power than the less curved: hence if parallel rays fall upon the surface the vertical rays will come to a focus sooner than the horizontal. The rays after refraction will be perfectly symmetrical when referred to the vertical and horizontal planes. They will have two foci. The whole bundle of rays is called Sturm's conoid, and the distance between the two foci is called the focal interval of Sturm. It is difficult to represent this conoid on a plane surface (Fig. 41), but we can see what sections of the bundle or pencil of rays would look like at different distances from the refractive surface (Fig. 41, A—G).

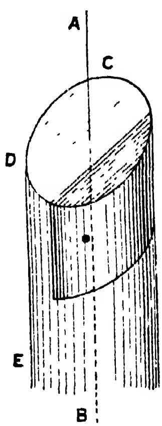


FIG. 42.

At A the section will be a horizontal oval or oblate ellipse, because the vertical rays are converging more rapidly than the horizontal. At B the vertical rays have come to a focus, while the horizontal are still converging: the section will be a horizontal straight line.

At C, D, and E the vertical rays are diverging and the horizontal are still converging. At one place in this focal interval there will be a spot (D) where the vertical rays have diverged from the axis exactly as much as the horizontal rays have converged towards it. Here the section is a circle, which is called the *circle of least diffusion*. At F the horizontal rays come to focus while the vertical are diverging: the section will be a vertical straight line. Beyond this point, as at G, both sets of rays are diverging, and the section will always be a vertical oval or prolate ellipse.

What will happen if the retina is situated at either of these points of section? In the first place it is obvious that the retinal image will always be blurred, and it is because the rays never come to a focus in a single point that the condition is called astigmatism (ἀ, privative, στίγμα, a point). If the retina cuts the conoid at A, where none of the rays have come to a focus, every meridian will be in the same condition, though in different degree, as in the axial hypermetropic eye: this condition is therefore called *compound hypermetropic astigmatism*. If the retina is at B the vertical meridian will be in the condition of an emmetropic eye, while the horizontal will still be in the condition of a hypermetropic one: this condition is called *simple hypermetropic astigmatism*. At C, D, and E the vertical meridian will be in the condition of a myopic, and the horizontal still in that of a hypermetropic eye: this is called *mixed astigmatism*. At F the vertical meridian is still myopic, whilst the horizontal is in the same condition as in an emmetropic eye: this is *simple myopic astigmatism*. Beyond F, as at G, both meridians are in the condition of an axial myope, the rays having crossed in the vitreous: this is *compound myopic astigmatism*. All these positions of the retina are met with in actual practice, though there is often a combination of axial and curvature defects.

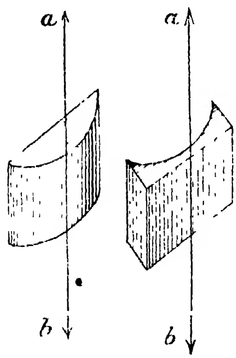


FIG. 43.

Distant vision is often found to be surprisingly good with relatively high degrees of mixed astigmatism, probably because the circle of least diffusion falls on or near the neuroepithelium of the retina.

It will be readily seen that such a condition cannot be corrected by means of any spherical lens. We must obtain some means of affecting one set of rays more than the other. This means is found in cylindrical lenses.

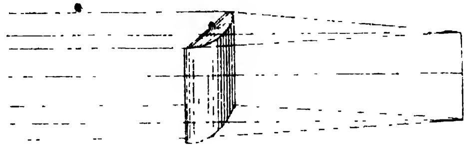


FIG. 44.—Refraction of parallel rays through a plano-convex cylinder.

Suppose C D E F is a cylinder of glass (Fig. 42): AB is called the axis of the cylinder. If a slice is cut off the cylinder by a plane parallel to the axis, it would form a cylindrical lens. Fig. 43 gives representations of a convex and a concave cylinder. The direction *ab* is called the axis of the cylinder, since it is parallel to the axis of the original cylinder from which the slice may be supposed to have been taken. It is important here to warn the student not to confuse the axis of a spherical and the axis of a cylindrical lens, as they are totally different things. The axis of a cylinder has just been described: the axis of a spherical lens is the line joining the centres of curvature of the two surfaces.

How will a cylindrical lens affect parallel rays falling upon its surface? In the direction of its axis it is simply a plane lamina with parallel sides, so that it will have no effect upon the rays. In the direction at right angles to its axis it is spherical on one side and plane on the other: it will therefore act exactly like a plano-convex or a plano-concave lens, *i.e.*, it will make the rays either converge or diverge. If a convex cylinder is held between a point of light and a screen, a position can be found for the screen such that a sharp bright line is thrown upon it (Fig. 45): this is the focal line of the cylinder.

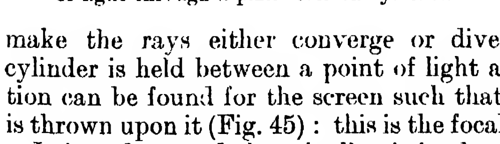


FIG. 45.—Refraction of divergent rays from a point of light through a plano-convex cylinder.

It is to be noted that the line is in the direction of the axis of the cylinder. If another convex cylinder of the same strength were held with its axis at right angles to the first, it would obviously form a focal line perpendicular to the first focal line. If the two cylinders are put in contact with their axes at right angles, all the rays after refraction must pass

through both lines. The only place where they can go through both lines is where the lines intersect. Hence we see that two cylindrical lenses of equal strength, placed in contact with their axes at right angles, act exactly like a convex spherical lens of the same strength as either of the cylinders.

When the cornea has its directions of greatest and least curvature at right angles to one another, the condition is called *regular astigmatism.* In the commonest form, as we have said, the vertical meridian is the more curved, the horizontal the less: this condition is generally called regular astigmatism "according to the rule." Sometimes the reverse is found: this is said to be "against the rule." Not infrequently the axes are oblique. Often after ulceration, &c., the surface of the cornea is irregular. This causes the rays of light

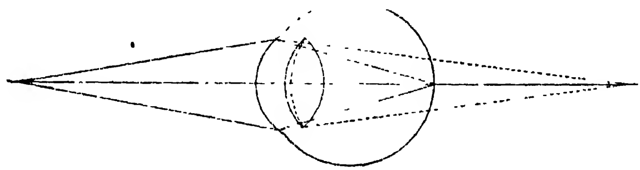


FIG. 46.—Effect of accommodation. The dotted lines show the curvature of the anterior surface of the lens and the course of rays with the eye at rest (static refraction). The solid lines show the curvature of the anterior surface of the lens and the course of rays with active accommodation (dynamic refraction).

to be refracted irregularly, so that there is no symmetry at all about them and different groups form foci in various positions. This is called *irregular astigmatism:* it cannot be corrected, and can only occasionally be improved by glasses.

Accommodation. We have to consider now how it is that a person with normal sight can not only see distant objects, but also near ones. If an object is situated near the eye, *e.g.*, at ordinary reading distance—about 22 cm. or 9 inches—the divergence of the rays which it emits cannot be neglected. Since the converging power of the refractive media of the emmetropic eye is only strong enough to make parallel rays come to a focus on the retina, it is obvious that divergent rays falling upon the cornea will not nearly have come to a focus (Fig. 46). They will indeed be made convergent, but only to such a degree that they would meet somewhere behind the retina. Now if we can make the converging power of the eye stronger, a point may be reached when it is just strong enough

to bring them to a focus on the retina. This is what is done by accommodation, and the manner in which the converging power of the eye is increased is by making the crystalline lens stronger.

We have seen that the refractive power of a convex lens depends upon its refractive index and upon the curvature of its surfaces. In accommodation, it is the latter which undergoes change. The curvature of the surfaces of the lens at rest in the eye is approximately spherical, and the radius of curvature of the anterior surface is 10 mm., while that of the posterior surface is 6 mm. In accommodation, the curvature of the posterior surface remains almost the same, but the anterior surface changes so that in strong accommodation its radius of

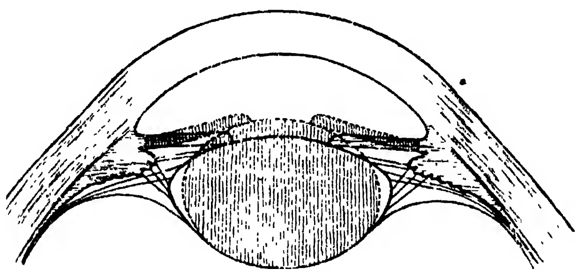


FIG. 47.—Diagram of Helmholtz' theory of accommodation.

curvature becomes 6 mm. The eye under these conditions, which are called its dynamic refraction, has a much increased converging effect upon the incident rays.

The mechanism by which this change in the curvature of the lens is brought about is as follows (Fig. 47). The lens substance is plastic, so that it tends to conform accurately to the shape of its capsule. As has been described (p. 10), the capsule is anchored to the ciliary body by the suspensory ligament. In the condition of rest, the fibres of the suspensory ligament are stretched. In the ciliary body is the ciliary muscle, which takes its origin from its anterior attachment to the sclerotic at the angle of the anterior chamber. When the muscle contracts, it pulls the posterior part of the ciliary body and the anterior part of the choroid forwards slightly. The effect upon the suspensory ligament is to slacken it, and with it the lens capsule. The posterior surface of the lens is fixed by the support of the jelly-like vitreous, so that the slackening of the

capsule makes itself most felt in the anterior part, which becomes bowed forwards. Since the anterior capsule is thicker behind the iris than in the pupillary area (Fig. 48) there is a nipple-like bulging of the lens through the pupil (Fincham).

The generally accepted theory here described is that of von Helmholtz. According to Tscherning the ciliary muscle tightens the suspensory ligament, so that the peripheral parts of the anterior surface of the lens are flattened and the central or pupillary area is increased in curvature.

Our control over the ciliary muscle, though involuntary, is very delicate, so that all distances up to quite close to the eye can be accurately focussed. The nearest point at which small

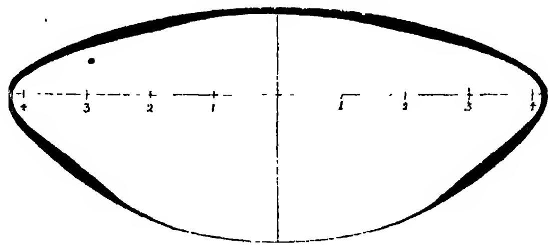


FIG. 48.—Lens capsule constructed from post-mortem specimens. Relative thickness magnified 100 times (Fincham).

objects can be clearly distinguished is called the *near point*, or *punctum proximum*. It is most accurately determined by gradually bringing a fine thread closer to the eye until it is found impossible to see it perfectly clearly. In practice it is sufficient to use very fine print and to determine the point at which it first becomes impossible to read it. The distance can be measured by a centimetre or inch tape held close beside the eye. For ordinary clinical purposes it is usually sufficient to judge the distance. At this point accommodation is exerted to its maximum, the lens capsule is as slack as it is possible to make it, and an object could only be seen clearly at a nearer point by placing a convex lens in front of the eye.

It has been shown that the far point of the eye varies according to its static refraction, *i.e.*, according to whether it is emmetropic, hypermetropic, or myopic. The near point also varies with the static refraction, but it also varies with the age of the patient, the reason being that the lens becomes less

plastic as age advances. We have stated that the lens is a mass of epithelium of which the central part is the oldest (*vide* p. 9). As the lens gets older the central cells become tougher and more compressed, thus forming a relatively hard nucleus. The nucleus is less plastic than the younger cortex, and as age advances more and more of the fibres become converted into nucleus. Consequently the lens tends less and less to respond to changes in tension of the capsule. Thus, a child of ten, the earliest age at which it is possible to obtain satisfactory measurements, is able to see a small object clearly when it is only 7 cm., or less than 3 inches, from the eye. A person of thirty years of age cannot see clearly at less than 14 cm., or about $5\frac{1}{2}$ inches, from the eye.

Now, we have pointed out that the refractive power of a lens in dioptres is the reciprocal of its focal distance measured in metres (*vide* p. 32). The same method is applied to measure the static and dynamic refractive powers of the eye. Thus, the static refractive power of a myopic eye whose far point is 1 metre in front of the eye is said to be 1 dioptre; this is usually expressed by saying that the eye has 1 D of myopia. Similarly, if a hypermetropic eye has its far point half a metre behind the eye it is said to have 2 D of hypermetropia. By this method the emmetropic eye, which has its far point at infinity, has no refractive power when it is at rest, since

$$\frac{1}{\infty} = 0.$$

Applying the same method to the dynamic refractive power, the child of ten, whose near point is 7 cm. from his eye, has a refractive power of $\frac{100}{7} = 14$ D, and a man of thirty, whose near point is 14 cm. from his eye, has a refractive power of

$$\frac{100}{14} = 7 \text{ D}.$$

By this means we can obtain a general rule for indicating the amount or *amplitude of accommodation*, not only of emmetropic but also of hypermetropic or myopic eyes. This is given by the formula $A = P - R$, which states that the amplitude of accommodation is equal to the refractive power of the eye when fully accommodated (*i.e.*, the reciprocal of the distance of the near point in metres) less the refractive power of the eye at rest (*i.e.*, the reciprocal of the distance of the far point in metres).

A few examples will make this clearer. Thus, the emmetropic child of ten has an amplitude of accommodation of $\frac{100}{7} - \frac{1}{\infty} = 14 - 0 = 14$ D. What is the amplitude of accommodation of an emmetrope whose near point is 12·5 cm. from his eye? Here $A = \frac{1000}{125} - \frac{1}{\infty} = 8$ D. From statistics which have been collected we can deduce that this man was about twenty-six years old (*vide* p. 54). Now let us take a case of myopia, *e.g.*, a myope of 2 D whose near point is 8 cm. in front of his eye. His amplitude of accommodation will be $A = \frac{100}{8} - 2 = 10·5$ D. What is the amplitude of accommodation of a hypermetrope of 3 D whose near point is 12·5 cm. from his eye? Here the far point is behind the eye and distances measured in this direction must have the opposite sign to those measured in front of the eye. Hence, $A = \frac{1000}{125} - (-3) = 8 + 3 = 11$ D.

The numbers given by these calculations for the amplitude of accommodation give the strength of the convex lens which would have to be placed in contact with the cornea in order that the near point might be brought to the required distance without using the accommodation. Several interesting facts come to light from the calculations. Thus a hypermetrope of 3 D has to exert 11 D of accommodation in order that he may see clearly at 12·5 cm., while an emmetrope has to exert only 8 D of accommodation to bring about the same result. We see, then, that the hypermetrope has to exert an amount of accommodation equivalent to the amount of his hypermetropia in order to focus parallel rays upon his retina, *i.e.*, he has to use this amount of accommodation in order to see distant objects clearly. Again, in the case of the myope of 2 D, his far point is half a metre, or 50 cm., from his eye; he can see clearly at that distance without accommodating, but he has to exert 10·5 D of accommodation in order that he may see clearly at 8 cm. from his eye. This patient, then, has to exert nearly as much accommodation to alter his points of clear vision from 50 cm. to 8 cm., *i.e.*, through 42 cm., that a hypermetrope of 3 D has to employ in order to move his point of distinct vision from infinity up to 12·5 cm. We see, therefore, that the *range of accommodation*, *i.e.*, the distance between the

far point and the near point, is not always the same for a given amplitude.

The effect of age upon the static and dynamic refraction is given in Fig. 51, which is the result of a large number of statistics and gives the average results. From this table we see that even the far point alters in advanced age. After about fifty the eye tends to become hypermetropic, so that at eighty it has about 2·5 D of hypermetropia. This has nothing to do with accommodation, and hence nothing to do with loss of plasticity in the lens. It is, however, due to changes going on in the lens, viz., an alteration in its refractive index so that it has a weaker converging power.

The refractive indices of the successive layers of the lens increase from the periphery towards the nucleus. The effect is twofold: it tends to correct aberration by increasing the convergence of the central rays, and the total refractive index of the whole lens is increased, being greater than the refractive index of the nucleus. For the lens may be looked upon as a central bi-convex lens encapsuled in two menisci (Fig. 49). The menisci act as concave lenses because the curvature of the nucleus is greater than that of the periphery of the lens. Hence they tend to counteract the effect of the central lens, but not so much as if their refractive index was the same.

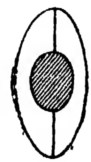


FIG. 49.

In old age the index of the peripheral layers usually increases, so that the total refractive index of the lens becomes less, and the eye becomes hypermetropic (*cf.* p. 313).

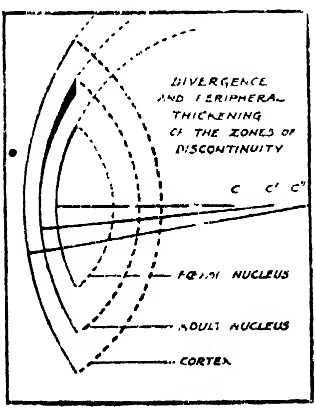


FIG. 50.

Examination with the slit-lamp (*vide* p. 97) reveals three chief surfaces of specular reflection corresponding to the anterior surface of the lens, the surface of the "adult nucleus," and the surface of the "fœtal nucleus" (Fig. 50) separated by "zones of discontinuity." The fœtal nucleus corresponds to the lens at birth. Its centre, the "central interval," is most homogeneous, and therefore appears darkest (*vide* Fig. 75, p. 98). The adult nucleus corresponds to the size of the lens in early adult life. It is separated from the lens capsule by the cortex, consisting of

lens fibres laid down subsequently (*vide* p. 9). Note that the radii of curvature of the successive bands diminish from without inwards, so that they act as negative menisci.

If we turn our attention to the curve of the near point we see that the amplitude of accommodation gradually diminishes throughout life. Now, we are accustomed to hold books for reading or work for sewing, &c., at about 10 inches, or 22 cm.

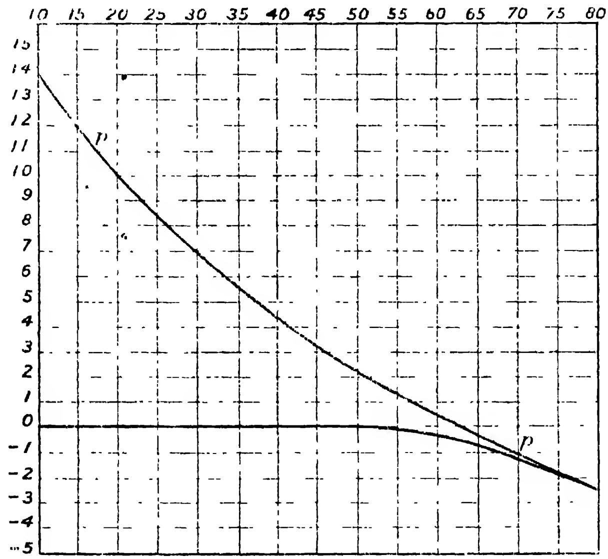


FIG. 51.—Chart of static and maximum dynamic refraction at various ages. (Donders.) Abscissæ, ages; ordinates, accommodation in dioptres.

from the eyes. In order to be able to see clearly at 25 cm. from the eye we have to exert $\frac{100}{25} = 4$ D of accommodation. If we look at the table we shall see that an emmetrope has only 4 D of accommodation left at about forty-one years of age. He will still be able to see clearly at 25 cm., but not closer. If he is about forty-six he will have only 3 D of accommodation left. He will not now be able to see clearly at 25 cm., but he will have to hold his book farther off, viz., at $\frac{100}{3} = 33$ cm. If he is still older he will have to hold the book yet farther off, and he will probably have to use very

large print or he will not see clearly at all. This is the condition which is called *presbyopia* (πρέσβυς, old).

It will be seen that a patient never requires more than + 4·D to correct his presbyopia only, since that is the amount of accommodation required to place the far point of the resting emmetropic eye at reading distance. A convenient rule to remember is that a presbyope may require + 1 D for every five years after forty; *i.e.*, at forty-five, + 1 D; at fifty, + 2 D; at fifty-five, + 3 D; at sixty and later, + 4 D. The rule errs in giving too liberal a correction; a smaller correction is often more comfortable, and owing to considerable individual variation each case must be treated on its merits.

It is a common error among students to think that presbyopia is a condition which commences at about forty-five years of age in emmetropes, and earlier in hypermetropes. Of course this is not so: the condition which has been increasing throughout life first becomes troublesome when the near point of the eye has receded so far that it is beyond comfortable reading distance.

There are two other phenomena which occur with accommodation, one affecting the iris, the other the direction of the eyes. In order that we may see a near object we must look at it; hence in order that we may see it at the same time with both eyes they must each turn inwards or converge. The amount of convergence, like the amount of accommodation, depends upon the distance of the object. It is therefore easy to understand that there is a close relationship between accommodation and convergence. We shall have more to say upon this subject when we consider the various forms of squint.

When we accommodate for a near object the pupil becomes smaller, or contracts. Experiment has shown that this movement of the iris is associated with the accompanying act of convergence rather than with accommodation *per se*. It is probably of the nature of an associated movement, or, as I have termed it, synkinesis (σύν, with, κίνησις, movement).

Contraction of the pupil during accommodation is not for the purpose of diminishing aberration, since this is already diminished by the act of accommodation. It has the effect of compensating for the relative increase of light entering the eye from near objects, but is greater than is necessary to produce this result.

It is not uncommon for the refraction of the two eyes to be

different: this condition is called *anisometropia* (ἀ, privative; ἴσος, equal; μέτρον, measure). It might be anticipated that this could be corrected to some degree or entirely by unequal accommodation in the two eyes. Thus, if one were emmetropic and the other hypermetropic, both eyes would be able to distinguish distant objects clearly if the hypermetropic one alone accommodated the requisite amount to correct its hypermetropia. It has been conclusively proved, however, that this does not occur. When these cases are not corrected by the proper glasses clear vision is wholly uniocular.

Although astigmatism is chiefly due to faulty curvature of the cornea, in some cases there is also lenticular astigmatism. This is not generally due to unequal curvature of the surfaces, but to slight tilting of the lens, so that the incident rays fall upon it obliquely. If we look through a tilted glass lens at printed matter we shall see that the letters become distorted and elongated in one direction; this is a form of astigmatism. The astigmatism of the crystalline lens is generally of such a nature that it tends to counteract the corneal astigmatism, though sometimes it adds to the effect. As in anisometropia, it might be thought that astigmatism could be corrected by accommodation. If, for instance, the ciliary muscle acted only at the sides and not at all above or below, the anterior surface of the lens would become more curved in the horizontal than in the vertical meridian. This would counteract the effect of the ordinary form of corneal astigmatism. It has been proved, however, that this also does not occur. When the ciliary muscle acts, it acts equally all round the circle; and when one ciliary muscle acts, the one in the opposite eye acts simultaneously and equally under ordinary conditions.

The Pupils

The iris acts like the diaphragm of any ordinary optical system, such as a photographic camera or a microscope. In discussing the effects of spherical mirrors in reflecting, and of spherical surfaces in refracting the rays of light, we said that in each case they were all brought to a focus in a single point. This is really only an approximation, which is sufficiently accurate for rays close to the axis. In a convex spherical lens, for instance, parallel rays near the axis meet at the principal focus (*vide* p. 29). The rays farther away from the axis, however, are refracted too much, so that they cut the axis nearer the lens than the principal focus (Fig. 52). This causes a

blurring of the edges of the image, which is said to be due to *spherical aberration.* The diaphragm cuts off these peripheral rays, and thus prevents the blurring. In the eye the surfaces are not even spherical near the periphery, and are often not so in the centre, so that much more aberration is liable to occur. The iris reduces the effects of the evil to a minimum.

There is also another form of aberration due to the imperfect refraction at spherical surfaces. White light is made up of all the colours of the spectrum. The component rays are refracted differently, the violet most, the red least. Hence there is a tendency for the white light to be split up into its components, in which case the image will have a coloured edge. This phenomenon is called *chromatic aberration.*

When light enters the eye and falls upon the retina the pupil

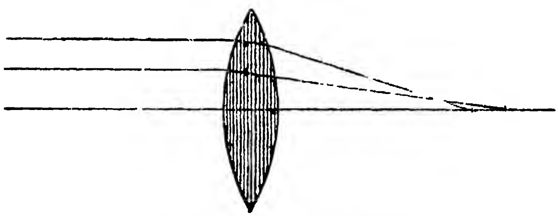


Fig. 52.—Spherical aberration.

contracts. We have already seen that the pupils also contract when the optic axes converge in accommodation. On the other hand, they dilate if the skin of any part of the body is pinched or any sensory nerve is stimulated to the extent of causing pain. These responses to stimuli of various kinds are very rapid and delicate, and are easily observed. When they are altered by disease the changes which occur afford very valuable information as to the condition of the nerve tracts involved. The tracts are rather complicated, but it is essential that they should be understood.

Under normal conditions, with equal illumination—a point too frequently neglected—the pupils are equal on the two sides. It is rare to meet with unequal pupils in a normal person; such cases do apparently occur, but every possible pathological cause must be eliminated before we admit that the condition is an idiosyncrasy.

On the other hand, the size of the pupils varies much in different people under the same conditions of illumination, &c. In old people it is smaller than in the young, sometimes to

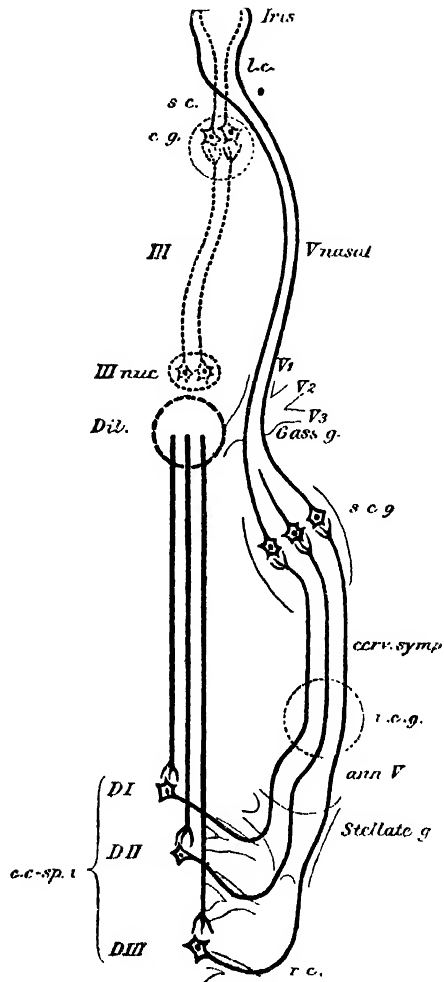


FIG. 53.—Diagram of the efferent pupillary paths. Dotted lines, pupillo-constrictor: *III nuc.*, nucleus of third nerve; *c.g.*, ciliary ganglion; *s.c.*, short ciliary nerves. Solid lines, pupillo-dilatator: *Dil.*, hypothetical dilatator centre in the medulla; *c.c-sp.i.*, Budge's centrum cilio-spinale inferius; *D.I*, *D II*, *D III*, first, second, and third dorsal nerves (see text); *r.c.*, ramus communicans; *Stellate g.*, stellate ganglion; *ann. V.*, annulus of Vieussens; *i.c.g.*, inferior cervical ganglion; *cerv. symp.*, cervical sympathetic; *s.c.g.*, superior cervical ganglion; *Gass. g.*, Gasserian ganglion; *V1*, *V2*, *V3*, first, second, and third divisions of the fifth nerve; *V nasal*, nasal branch of the ophthalmic (first) division of the fifth nerve; *l.c.*, long ciliary nerves.

so great an extent that the pupils are almost "pin-point." They are often smaller in hypermetropes, and larger in myopes, than in emmetropes; they are said to be smaller in blue eyes than in brown. The causes of these differences are conjectural, and need not detain us. The two facts of prime importance have been mentioned, viz., that there are two reflexes, that to light and that to sensory stimulation, which act in opposite directions. The normal size of the pupil may be looked upon as essentially the resultant of these two forces.

The motor innervation of the pupil is as follows (Fig. 53). The sphincter pupillæ is innervated by the third cranial nerve. The pupil-constrictor fibres start in the anterior part of the third nucleus in the floor of the aqueduct of Sylvius. They pass out of the mid-brain and run in the main trunk of the third nerve as far as the orbit. Here the fibres pass into the branch which supplies the inferior oblique muscle, leaving it by the short root of the ciliary ganglion. From the ciliary ganglion they pass by the short ciliary nerves to the eye, piercing the sclerotic around the optic nerve, being here in company with the short ciliary arteries (*vide* p. 11). The nerve fibres pass forwards in the choroid and ciliary body to the iris.

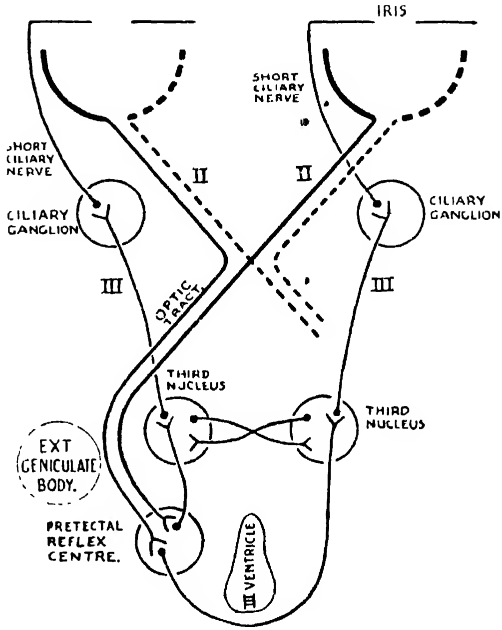


FIG. 54.—Diagram of the afferent and efferent pupillary paths for light stimuli. Afferent paths from left sides of retinæ, thick solid lines; afferent paths from right sides of retinæ, thick dotted lines; efferent paths, thin solid lines. *II.*, optic nerve. *III.*, third nerve.

The dilatator pupillæ is supplied by the cervical sympathetic

nerve. The dilatator tract probably commences in the mid-brain not far from the constrictor tract. It passes downwards through the medulla oblongata into the lateral columns of the cord. The fibres leave the cord by the ventral roots of the first three dorsal and probably the last two cervical nerves, enter the rami communicantes, and run to the first thoracic or stellate ganglion. From here they pass by the anterior limb of the annulus of Vieussens into the cervical sympathetic. In this nerve they run up the neck to the superior cervical ganglion, whence they pass with the carotid plexus into the skull. They run over the anterior part of the Gasserian ganglion and pass into the first or ophthalmic division of the fifth nerve, following the nasal branch, which they leave finally to enter the long ciliary nerves, thus avoiding the ciliary ganglion. The long ciliary nerves enter the eye on each side of the optic nerve, accompanying the long ciliary arteries. Like them, they run forwards between the choroid and sclerotic, enter the ciliary body and thus reach the iris.

These complicated paths will be seen at a glance in the accompanying diagrams (Figs. 53, 54).

We have now to consider the nervous mechanism of the reflexes. The light reflex is carried out entirely through the constrictor centre, so we may put the dilatator tract aside for the moment. The afferent fibres are contained in the optic nerve, arising from all parts of the retina (Fig. 54). It is unknown whether they belong to the large or the small fibres of the optic nerve. It is certain that they undergo partial decussation in the chiasma, like the visual fibres, and that they enter the optic tracts. It is also certain that, unlike the visual fibres, they do not enter the lateral geniculate body, but leave the tract to enter the pretectal region; thence the fibres from each tract are relayed to both third nuclei (Fig. 54).

The constrictor centre possesses "tone," *i.e.*, it is perpetually sending out impulses which keep the pupil slightly contracted. If light falls upon the retina of one eye its pupil contracts—*the direct reaction to light;* but the pupil of the opposite eye also contracts simultaneously—*the consensual reaction to light.* This consensual reaction should always be tested, since it gives useful information which cannot be obtained from the direct reaction. Thus, if there is a block on one optic nerve so that there is no direct reaction to light, but the consensual reaction from light thrown upon the other eye is unimpaired, we know that the block, whatever it may be,

does not affect the efferent constrictor tract, *i.e.*, the trunk of the third nerve, the branch to the inferior oblique, and the short ciliary nerves are intact. The consensual reaction is probably carried out by means of fibres which unite the two constrictor centres in the third nucleus.

That the afferent pupil-constrictor fibres undergo partial decussation in the optic chiasma is proved by Wernicke's *hemianopic pupil reaction.* This reaction is pathognomonic of disease of one optic tract. It will be seen from Fig. 54 that such a lesion will cut off the afferent impulses from corresponding halves of each retina, *i.e.*, from the temporal half of one and the nasal half of the other. If light is thrown upon these parts of the retinæ the pupils do not contract, but if it is thrown on the other halves of the retinæ the pupils respond.

The sensory reflex is more complicated than the light reflex, for both the dilatator and the constrictor centres play a part in its production under normal conditions. It has been shown that sensory stimulation causes first a rapid dilatation of the pupil due to augmentation of the dilatator tone through the cervical sympathetic, and then a second dilatation, rapid in onset but slow in disappearance, due to inhibition of the constrictor tone. There are other reflexes and synkineses, *e.g.*, emotional, which need not detain us.

Minute examination of the pupil when the intensity of the light entering the eye is altered, shows that the pupil contracts and then oscillates rapidly, finally settling down into a condition of contraction which is slightly less than the summit of the first wave. In its sudden response, the pupil as it were oversteps the mark, oversteps it again in the opposite direction, and so on. Two different types of exaggeration of this oscillation are met with under abnormal conditions. One is the condition in which the oscillations are very large and easily seen, and which are to a large extent independent of the light falling upon the eye. This is called *hippus*; its origin is obscure, but it undoubtedly depends upon the rhythmic activity of the nervous centres, and is not a peripheral phenomenon. More important is the lack of sustained contraction under the continued influence of light. Here the pupil contracts sluggishly when the intensity of the light is increased, but while the light is still kept constant it slowly dilates, often with superposed sluggish oscillations. This is a pathological phenomenon dependent upon diminished conductivity in the afferent path of the light reflex, *i.e.*, usually in the optic nerve (*see* Retrobulbar Neuritis).

Drugs are so frequently employed in ophthalmic practice for the purpose of dilating or constricting the pupils or paralysing the accommodation, that it is important to know exactly how they act. Pupil-dilating drugs are called mydriatics; pupil-constricting, miotics; drugs which paralyse the ciliary muscle, cycloplegics. All drugs which dilate the pupil also paralyse the accommodation in greater or less degree; many attempts have been made to discover a drug which will effect the former purpose without the latter, but without success. Similarly, all miotics stimulate the ciliary muscle to contract, so that the eye assumes a condition of partial or complete accommodation.

Most of these drugs do not apparently act directly on the muscles or on the nerve-endings. Constriction of the pupil by the third nerve is due to the liberation of acetylcholine (Englehardt). Atropine destroys or prevents the formation of this substance; hence it can only be counteracted by substances which act directly on the muscle, *e.g.*, histamine. Eserine acts by preventing the normal rapid destruction of acetylcholine, hence the extreme irritability of the sphincter and ciliary muscle, and of the eye in bright light. Hence, eserine cannot counteract atropine mydriasis, whereas atropine easily counteracts eserine miosis.

The strongest mydriatic which we possess is *atropine;* it paralyses the sphincter iridis and ciliary muscle completely, and is said also to stimulate the dilatator iridis. It has so potent an action that it abolishes the tone of the ciliary muscle. Thus, an emmetropic eye placed fully under the influence of atropine becomes hypermetropic to the extent of about 1 D; this must be taken into account in correcting errors of refraction. Atropine solution (*e.g.*, 1 per cent.) instilled into the conjunctival sac is absorbed through the cornea into the anterior chamber, where it acts locally upon the intrinsic muscles. It takes a considerable time to cause complete paralysis, hence it is usual to order it for use at home three times a day for at least three days. The effects do not pass off for about ten days. One drop of 0·5 per cent. atropine sulphate solution causes wide dilatation of the pupil in thirty to forty minutes, and complete paralysis of accommodation in about two hours; the effects do not pass off entirely till from three to seven days. Duboisine, hyoscine or scopolamine, and daturine act similarly to atropine.

Homatropine acts more quickly than atropine, and the effects pass off more quickly. Its full effect is obtained by an oily

solution (in ol. ricini, 1 per cent.) in three-quarters of an hour, especially if combined with cocaine (2 per cent.), which acts chiefly by increasing the permeability of the cornea. The effects pass off completely in forty-eight hours, or much more quickly if a drop of eserine (1 per cent.) is instilled. The mixture of homatropine and cocaine, which is commonly employed for estimating refraction, does not paralyse the intrinsic muscles so fully as atropine, the tone of the ciliary muscle not being abolished so thoroughly. Homatropine probably acts upon the iris through the sphincter only.

Cocaine, besides its anæsthetic effect through the endings of the fifth nerve in the cornea, iris, &c., also stimulates the sympathetic nerve endings in the dilatator iridis. It does not paralyse the sphincter, so that the dilatation of the pupil is only moderate, and the pupil continues to react to light even after prolonged application. Cocaine is a useful drug in confirming the diagnosis of paralysis of the sympathetic nerve: if this nerve is paralysed cocaine fails to dilate the pupil. The effect is not due to degeneration of the nerve endings, as I have found that cocaine fails to act very soon after section of the sympathetic in the neck in animals.

Eserine, or physostigmine, the most powerful miotic we possess, acts by stimulating the third nerve endings in the sphincter and in the ciliary muscle. It is therefore an antagonist of atropine, but it is unable to overcome the dilatation produced by 1 per cent. atropine. On the other hand, eserine readily overcomes the dilatation produced by homatropine and cocaine. These facts are of very great importance and must be carefully borne in mind (*vide*, p. 261). Comparably with cocaine eserine fails to produce constriction of the pupil after section of the third nerve.

Eserine, unlike the common mydriatics, causes some smarting and injection of the ciliary vessels when instilled into the conjunctival sac. What is more unpleasant is the "dragging" sensation in the eye which patients complain of when it is acting. It may be so irritating as to cause vomiting, but this only occurs in very sensitive persons or when the drug is pushed. Owing to these symptoms it should not be instilled more frequently nor in stronger doses than requisite to ensure the desired result. A 0·5 per cent. solution or one considerably weaker is often adequate.

Eserine begins to contract the pupil and cause spasm of accommodation in about five minutes; its maximum effect is reached in twenty to forty-five minutes. The effect on

accommodation lasts only an hour or two, that on the pupil two to three days.

Pilocarpine causes miosis by directly stimulating the parasympathetic apparatus. The action is less prolonged and may be followed by a fatigue reaction—slight mydriasis.

In irritative miosis, due to stimulation of the third nerve, light, accommodation, and eserine will cause greater constriction, atropine dilatation. In paralytic miosis, due to paralysis of the sympathetic, light, accommodation, and eserine will cause constriction, atropine little or no dilatation.

In spastic mydriasis light, accommodation and eserine will cause constriction. In paralytic mydriasis there is no reaction to light or accommodation, and eserine acts very feebly.

Adrenaline causes dilatation of the pupil in cases of acute pancreatitis (Loewi): four drops of 1 in 1000 solution should be instilled into one conjunctival sac, and the instillation repeated in five minutes. The dilatation is manifest after half an hour, the pupil being often oval (Garrod).

Histamine ("amino-glaucosan") produces maximum miosis by acting directly upon the muscle fibres.

Visual Perceptions

When light falls upon the retina it acts as a stimulus to a sensory nerve ending. As contact of the skin with a foreign substance causes the sensation of touch, so stimulation of the retina causes visual perceptions. The changes which go on as the result of a suitable stimulus in an ordinary tactile end organ, the physiological impulses in the afferent sensory nerves, and the psychological interpretation of these impulses in the brain which we call tactile sensation, are all relatively simple. In the visual nervous mechanism they are much more complex and highly differentiated.

We may first very briefly consider the changes which occur in the end organ itself. Light falling upon the retina causes mechanical, photochemical, and electric responses. (1) The pigment in the hexagonal cells of the retinal epithelium migrates from the bodies of the cells into the processes which lie between the rods and cones. (2) The cones become shorter. Both these effects are slow; and it is doubtful whether these mechanical effects occur to an appreciable extent in primates and man. (3) The visual purple, a substance which is found only in the rods, is bleached, so that a sort of photograph or

optogram of the luminous object is formed. (4) Changes of electrical potential are set up in the retina.

We are more concerned, however, with the sensations which result from stimulation of the retina with light. These are of three kinds, which are called the Light Sense, the Colour Sense, and the Sense of Form. Each of these may become disordered, so we must examine what they really mean.

The *Light Sense* is the faculty which permits us to perceive light, not only as such, but in its gradations of intensity. By

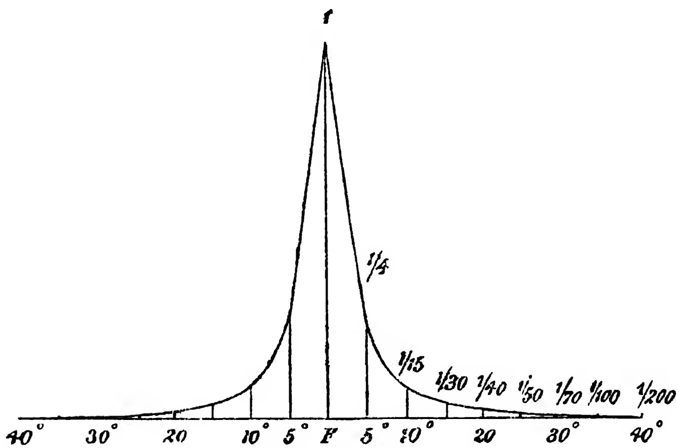


FIG. 55.—Acuity of form sense in different parts of the retina. (Dor.) *F*, fovea centralis. Abscissæ, degrees towards the periphery of the retina ; ordinates, relative acuity, that at the fovea being unity.

utilising shadows cast by the retinal blood vessels upon the rods and cones it can be proved that the neuro-epithelium is the actual sentient layer of the retina (Purkinje's experiment). It is in this layer that the clear images of objects in the outer world are focussed. The rays stimulate the rods and cones and give us the sensation of light. Hence rays falling upon the optic disc give rise to no visual sensation, and this is therefore called the *blind spot* (Mariotte).

If the light which is falling upon the retina is gradually reduced in intensity there comes a point when it is no longer perceived : this is called the *light minimum*. It varies very greatly according to the amount of light which has been falling upon the retina before the observation is made. We are all

aware that if we go from bright sunshine into a dimly lit room we cannot perceive the objects in the room until some time has elapsed: the eyes have to become "adapted" to the amount of illumination. Hence observations on the light minimum are only comparable one with the other when the eyes are in the same condition of dark adaptation. Since this involves keeping light from the eye for at least twenty to thirty minutes the investigation is tedious, and perhaps this is the chief reason why it has been much neglected clinically. The light minimum for the fovea is considerably higher than for para-central and peripheral parts of the retina, and retinal adaptation affects the macula relatively little.

Having ascertained the light minimum, if the light is gradually increased in intensity there are points at which we can clearly distinguish a difference in the amount of illumination. We can do this best if we have two illuminated areas of equal size to compare, as is done in special instruments for the purpose, called photometers. We can then find out how much brighter one area must be in order that we may distinguish a difference in illumination. This is called the *light difference.* It is found to vary with the amount of illumination—the greater the amount of illumination the greater will have to be the difference in order that we may be conscious of any difference at all. Indeed, light difference tends to follow a well-known law which is approximately valid for all sensory impressions (Weber's Law).

Neither of these functions of the light sense is much used in ophthalmology. There is no doubt that the light minimum is increased in diseases which impair the percipient elements, viz., the rods and cones (*see* Retinitis pigmentosa). It is said that disease of the conducting elements, the nerve fibres, causes increase in the light difference.

The rods are much more sensitive to low illumination than the cones, so that in the dusk we see with our rods (*scotopic vision*). Nocturnal animals, like the bat, have few or no cones.

The *Form Sense,* which is next in importance, is the faculty which enables us to perceive the shape of objects in the outer world. Here the cones play the predominant part, and where they are most massed together and most highly differentiated, viz., at the macula, there the form sense is most acute. It falls off very rapidly towards the periphery, as is shown in Fig. 55, and it is noticeable that the curve agrees fairly well with the diminution in the number of cones. We are accus-

tomed to speak of acuity in distinguishing the shapes of objects as *acuity of vision*, and we mean by that the greatest acuity which it is possible to obtain. The acuity of vision, therefore, applies to central vision, or the vision of objects whose images are formed at the fovea and its immediate neighbourhood, the macula lutea.

The form sense is not a purely retinal function, but in the perception of composite forms—such as letters—is largely psychological. A punctate source of light does not form a punctate retinal image, but a circle of diffusion. The size and definition of this depend upon the resolving power of the eye, regarded as an optical instrument and vary with the wave-length of the light, pupil aperture, etc. The purely physiological elements which enter into the complex form sense are (1) the light sense, (2) the sense of position, (3) the sense of discrimination. The sense of position depends upon the light sense and upon the conditions of contrast between the object and its background. A physiological process, called spatial induction, causes a lowering of sensibility of the area surrounding a stimulated area, so that the demarcation between the two areas is increased. The sense of position is measured by the *minimum visibile*. The sense of discrimination is the power to distinguish two visible objects as separate, and is measured by the *minimum separabile*. The finest sense of discrimination of any sensory organ is the visual capacity to distinguish an irregularity in the line of demarcation between two contours, which is of the order of a visual angle of 5 seconds of arc—much less than the so-called minimum visual angle (*v. infra*). It is the basis of the accuracy of physical measurements with the vernier. Form sense is measured by the *minimum legibile* or *cognoscibile*.

In determining the acuity of vision, we utilise the visual angle (*vide* p. 37). We naturally choose as our basis the *minimum visual angle*, *i.e.*, the angle which two luminous points must subtend at the nodal point of the eye in order that they may be perceived as separate and distinct. Now, in order that we may get separate impressions from two points close together on the retina it is necessary that two cones shall be stimulated, and that there shall be a cone between these two which is not stimulated. If we know the diameter of a cone we can calculate the minimum angle which must be subtended at the nodal point in order that these requirements may be fulfilled. This angle, as we have already seen, is equal to the angle subtended on the other side of the nodal point by the two luminous points. As a matter of fact these calculations

agree fairly well with the results of observations. It might be thought that the observation was a very easy one, but there are several complications. It is found that there is a certain amount of spread of the stimulus from one cone to surrounding ones, due to the size of the pupil, spherical and chromatic aberration, and irregular astigmatism of the refractive media, especially the lens; it is somewhat diminished by diffraction at the edge of the pupil. This causes bright objects on a dark background to appear a little larger than they really are: the phenomenon is called irradiation. It is not altogether a disadvantage, for it helps us very much in seeing actual points of intense light, like the stars, millions of miles away. In clinical work it would be a disadvantage to have an unduly high standard of visual acuity, because nearly everybody would be abnormal according to the standard. It is found that a minimum visual angle of 1 minute (1′) or one-sixtieth of a degree gives a very good average, and it is upon this basis that test types are constructed (*vide* p. 131).

The *Colour Sense* is that faculty whereby we are enabled to distinguish different colours and different colour tones. The exact investigation of the colour sense is one of great complexity, for the different colours of the spectrum differ in luminosity, so that this disturbing factor has to be added to those dependent upon the physiological condition of the retina, *e.g.*, its state of adaptation, and so on.

Appreciation of colours occurs only with lights of moderate or high intensity and some degree of light adaptation of the retina (*photopic vision*). If a spectrum of low intensity is viewed with the dark adapted eye it appears as a grey band, differing in brightness in different parts (*scotopic vision*). The brightest part is at about 510 $\mu\mu$, corresponding to the green of the photopic spectrum. The brightest part of the photopic spectrum is at about 555 $\mu\mu$, nearer the yellow. Scotopic vision is essentially a function of the rods, colour vision of the cones (duplicity theory).

If three colours sufficiently far apart in the spectrum are chosen, all the other colours, though not in the same degree of saturation, can be formed by their combination in suitable proportions, and white light can also be formed in the same manner. Hence normal colour vision is called trichromatic. There are reasons for choosing red, green and blue for these "primary" colours. Now, we know that physiological impulses are in some sense a reflex or image of the physical

stimuli which give rise to them, *e.g.*, sound waves cause physiological impulses, which are perceived as sound, and so on. If, therefore, we imagine three slightly different kinds of impulse set up by the stimulation of the retina by red, green and blue light respectively, their combinations in suitable proportions would enable us to perceive the whole gamut of the spectrum, including white light. This is the basis of the Young-Helmholtz theory of colour vision. According to Hering's theory, chemical changes in three different types of "visual substance" situated in the retina cause the sensations of colour (including white and black). The three substances are white-black, red-green, and blue-yellow. If anabolic or building-up changes (assimilation) are set up in these substances, the sensations of white, red and blue are caused respectively. If katabolic or breaking-down changes (dissimilation) are set up, black, green, and yellow result.

Perhaps no subject affords a better field for conjectures than the theory of colour vision. It is sufficient here for us to emphasise the importance of the three primary colours, upon which stress is laid by the Young-Helmholtz theory, and the intimate relations which exist between red and green, blue and yellow, and white and black, which are the foundation of the Hering theory. It is necessary to realise so much because the colour sense is defective in a fairly large proportion of people. This congenital abnormality is called *colour blindness* (*q.v.*). In it the importance of the three primary colours and the intimate relation of the pairs of colours are forcibly brought out.

The three types of visual perception are not confined to the minute area of central or macular vision. All are present in greater or less degree in more peripheral parts of the retina. In disease the earliest and most delicate traces of failing function are often to be found in the peripheral parts, central vision remaining perfectly normal. Hence the great importance of knowing the normal limits of the light perceptive and the colour perceptive areas of the retina.

The *field of vision* is the projection of these percipient areas of the retina on the outer world. When we stand upon the seashore and look at, or "fix," a ship on the horizon it forms a retinal image at the fovea. We are accustomed, then, to consider that any object in the outer world which forms the image at the fovea is situated somewhere upon the *line of vision*, *i.e.*, the line passing through the fovea and the nodal point of the eye. The foveal image is "projected" outwards along this line. Whilst still fixing the ship we are conscious of

seeing, less clearly, innumerable objects for miles around. From our knowledge of the refractive mechanism of the eye we know that these objects must form their retinal images upon peripheral parts of the retina. Regarded from the side of the eye the image upon any point of the peripheral part of the retina is "projected" outwards along the line joining the point with the nodal point. The field of vision, then, is the projection outwards of all the points upon the retina which can give rise to visual perceptions. We will postpone the consideration of its properties to a later stage (*vide* p. 138).

CHAPTER V

The Neurology of Vision

In the preceding chapter we have considered the process of vision up to the point at which the retinal receptive elements, the rods and cones, have become stimulated. As with other

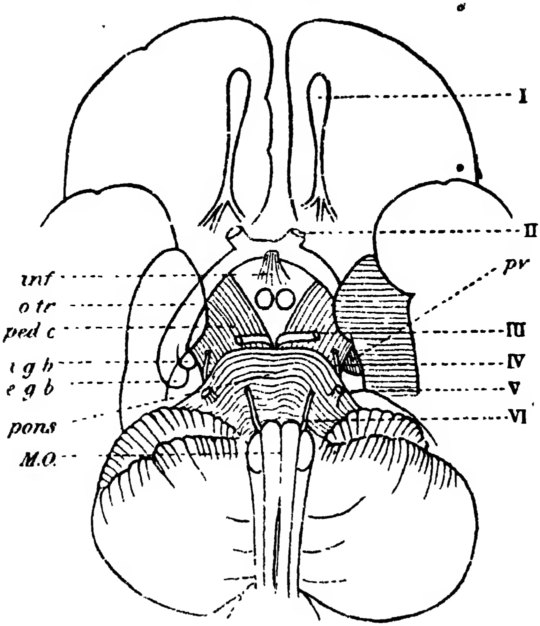


FIG. 56.—Diagram of the base of the brain, showing superficial origin of the I, II, III, IV, V, and VI cranial nerves : *inf.*, infundibulum ; *o.tr.*, optic tract ; *ped.c.*, cerebral peduncle ; *i.g.b.*, internal geniculate body ; *e.g.b.*, external geniculate body ; *pv.*, pulvinar of optic thalamus ; *M.O.*, medulla oblongata.

sensory nerves, stimulation of the end organ causes the development of nervous impulses which travel up the afferent tracts of the central nervous system to the brain. The comparison of the afferent tracts of common sensation with those of vision throws so much light upon the latter that it is worthy of a moment's consideration.

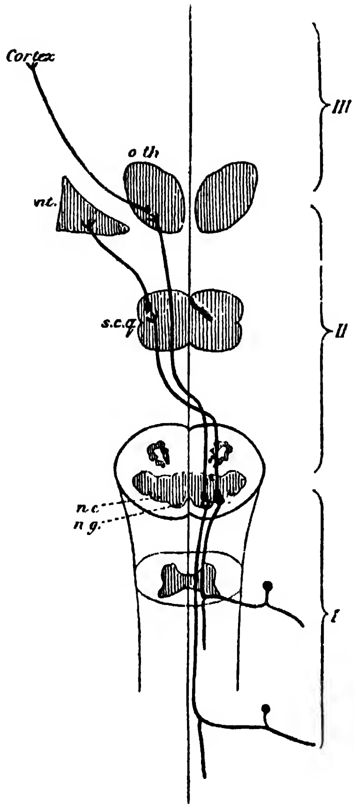


FIG. 57.

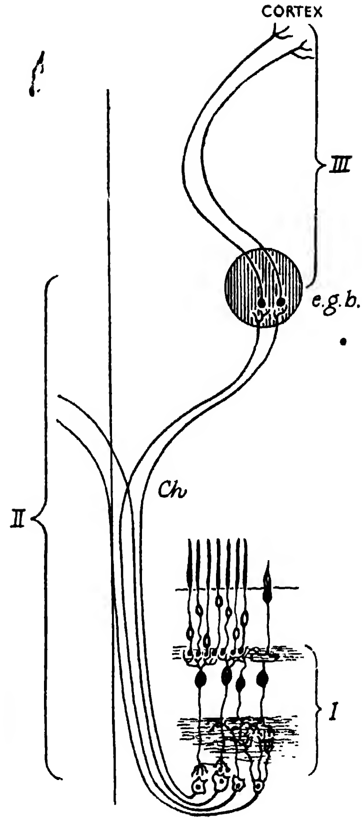


FIG. 58.

FIGS. 57 and 58.—Fig. 57 represents a diagram of the neurones of the most direct path of common sensation. Fig. 58 represents the neurones of the afferent visual path. I, II, III, neurones of the first, second, and third orders respectively; *n.c.*, nucleus cuneatus; *n.g*, nucleus gracilis; *s.c.q.*, superior corpus quadrigeminum; *n.lent.*, nucleus lenticularis; *o.th.*, optic thalamus. I in Fig. 58, rod and cone bipolars in the retina. *Ch.*, chiasma; *e.g.b.*, external geniculate body.

The sensory impulse of common sensation, *e.g.*, in the leg, is carried by a nerve fibre along the sensory nerve and the dorsal spinal root to the cord: it travels up in the posterior columns of the cord to the nucleus gracilis or the nucleus cuneatus as the case may be (Fig. 57). The whole of this course is along

the processes of a single cell or neurone, which has been called the neurone of the first order. The impulse is taken up in the nucleus gracilis or cuneatus by a second cell, and is carried along the nucleo-thalamic tract or mesial fillet to the opposite optic thalamus; other fibres, especially those derived from the nucleus cuneatus, pass to the superior colliculus or corpus quadrigeminum. The cells in the nuclei gracilis and cuneatus

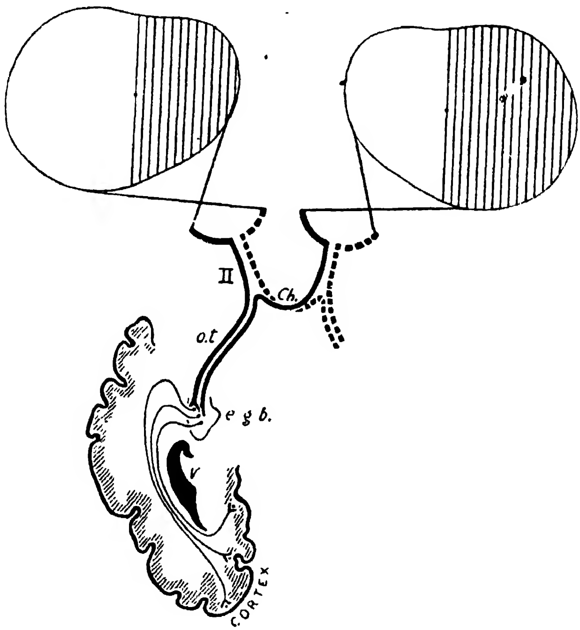


FIG. 59.—Diagram of the afferent visual paths from the retinæ, with the corresponding fields of vision. *II.*, optic nerve; *Ch.*, chiasma; *o.t.*, optic tract; *e.g.b.*, external geniculate body; *v.*, lateral ventricle.

are the neurones of the second order. A third cell, the neurone of the third order, situated in the thalamus or colliculus, carries on the impulse to the cortex cerebri. Here the nervous impulse is transformed into a psychic impulse, a change which is not and probably never can be understood.

Let us compare with this the visual afferent tracts (Fig. 58). The end organ is the neural epithelium of rods and cones. The first true conducting nerve cell or neurone of the first order is the bipolar cell of the inner nuclear layer with its axon in the

inner reticular layer. This microscopic cell corresponds morphologically with a dorsal root ganglion cell and its long processes stretching, in some cases, from the tip of the toe to the top of the spinal cord. The neurones of the second order are the ganglion cells in the retina whose processes pass into the nerve fibre layer and along the optic nerve to the lateral or external geniculate body. Here a new cell, the neurone of the third order, takes up the transmission of the impulse, travelling

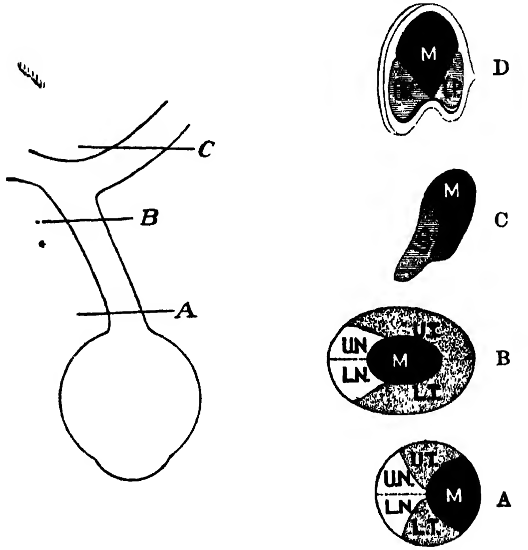


FIG. 60.—Distribution of fibres of the Optic Nerve. M., macular; U.T., upper temporal, L.T., lower temporal; U.N., upper nasal, L.N., lower nasal; U.P., upper peripheral, L.P., lower peripheral; A, B, in optic nerve; C, in tract; D, in external geniculate body. The right-hand portion of the figure is reproduced from Eugene Wolff's "Anatomy of Eye and Orbit," Lewis, London. (After Brouwer & Zeeman.)

by way of the optic radiations of Gratiolet to the cortex of the occipital lobe, which is the so-called *visual centre*.

Earlier observers recorded fibres passing from the tract to the pulvinar of the optic thalamus and to the superior colliculus. These have not been confirmed, but it is surprising that there is no connection with the latter, the phylogenetically primitive visual centre.

We see, then, the morphological identity of the two systems, in spite of the great anatomical differences which specialisation has brought about. We may emphasise again the fact that

the peripheral optic nerve proper is a bipolar cell in the inner nuclear and inner reticular layers of the retina, while the so-called optic nerve is a part of the central nervous system homologous with the mesial fillet in the medulla and pons.

We must now investigate more minutely the individual parts of the visual system. The results which are about to be described are derived from three sources—embryology, experiments upon animals, and clinical observation as controlled by post-mortem findings.

Let us first trace the fibres from the various parts of the retina (Fig. 59). In general it may be said that the fibres from peripheral parts enter the middle of the nerve, while the fibres from parts near the nerve enter the peripheral parts of the nerve: they probably maintain this relative position as far back as the chiasma. There is, however, one disturbing factor, viz., the fibres from the macular region. This part is specially well supplied. The fibres pass into the outer part of the nerve, where they are spread over an area which is triangular in section, with the apex towards the centre of the nerve (Fig. 60). These *papillo-macular fibres* soon become more centrally situated, so that in the posterior part of the nerve they are all in the centre. Tracing them still farther backwards the nasal fibres decussate in the chiasma, while the temporal ones enter the optic tract of the same side. They pass to the dorsal part of the lateral geniculate bodies (Le Gros Clark). The axons of their corresponding neurones of the third order are also widely distributed in the central part of the optic radiations and end at the most posterior part of the visual cortex at the tip of the occipital pole (Gordon Holmes and Lister); each half macula is thus represented in the opposite occipital lobe (Figs. 63, 64).

The consensus of opinion is now against the view that each macula is represented in each occipital lobe (*vide* p. 407).

The fibres from peripheral regions of the retina, similarly, form two distinct groups, corresponding with the temporal and nasal halves of the retina. The limitation is very exact, as if a vertical line divided the retina into two halves at the level of the fovea (Fig. 59). The fibres from the temporal half of the retina enter the chiasma but do not decussate; they pass into the optic tract of the same side. Thence they run to the lateral geniculate body where all the visual fibres end. The fibres from the nasal half of the retina enter the

chiasma, decussate, and pass into the optic tract of the opposite side having a similar distribution on the other side. The direct and crossed fibres pass to alternating laminæ in the lateral geniculate body (Minkowski, Le Gros Clark). The corresponding neurones of the third order pass by the optic radiations to the corresponding occipital lobes. We see, therefore, that a lesion of one occipital lobe or optic tract will cause blindness of the temporal half of the retina on the same side and of

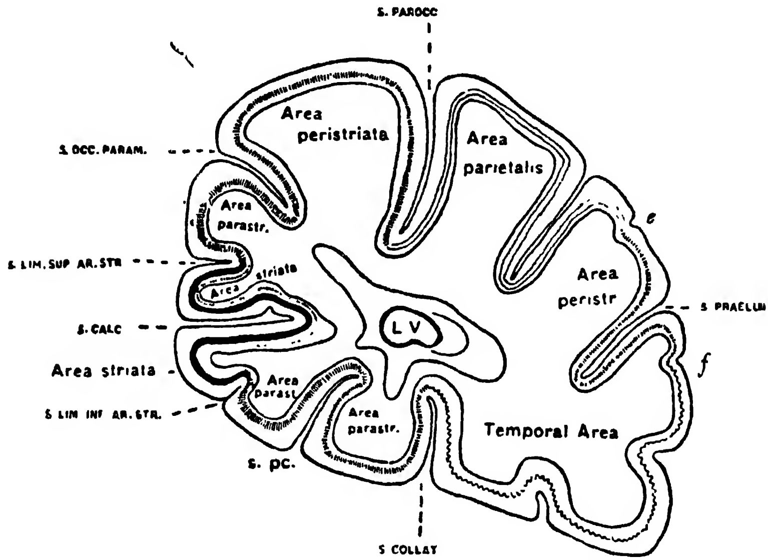


FIG. 61.—A diagram to represent the distribution of the different cortical areas in a coronal section through the left hemisphere one centimetre behind the fossa parieto-occipitalis. *LV.*, posterior cornu of lateral ventricle ; *s.calc.*, posterior calcarine sulcus ; *s.lim.sup.ar.str.*, sulcus cunei (sulcus limitans superior areæ striatæ) ; *s.lim.inf.ar.str.*, sulcus lingualis (sulcus limitans inferior areæ striatæ). Note how the stripe of Gennari (the thick black line) ends abruptly at the bottom of the sulcus cunei and the sulcus lingualis. (After Elliot Smith.)

the nasal half of the retina on the opposite side. Projecting this outwards, such a lesion will cause loss of vision in the opposite half of the binocular field of vision, a condition which is known as *hemianopia* (hemiopia, hemianopsia). We may recall the fact that the afferent pupil-constrictor fibres have a similar semi-decussation in the chiasma.

The visual fibres in the optic radiations maintain a posterior position in common with the sensory fibres from the rest of the

body, being thus situated behind the motor fibres in the internal capsule. Thereafter they separate considerably, the ventral fibres (projecting the lower quadrant of the retina or the upper quadrant of the visual field) running forwards far into the temporal lobe before they turn backwards to the lower portion of the visual cortex, the dorsal fibres (projecting

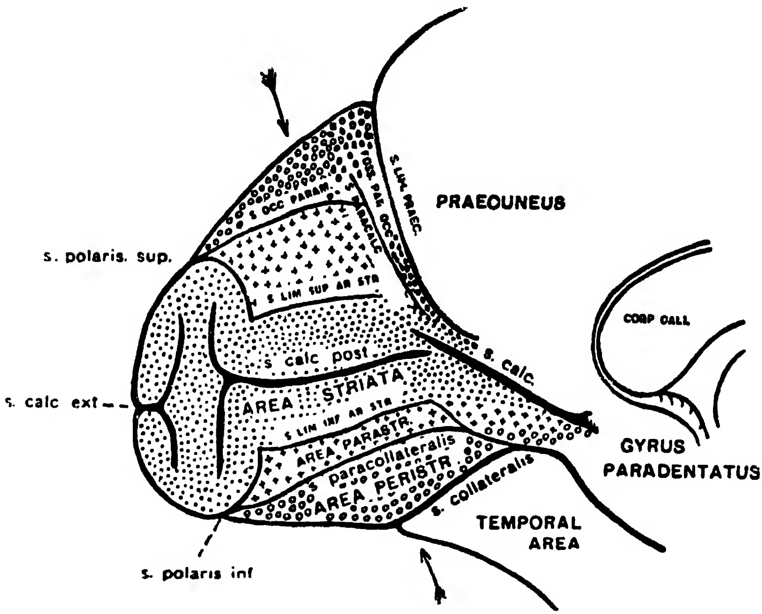
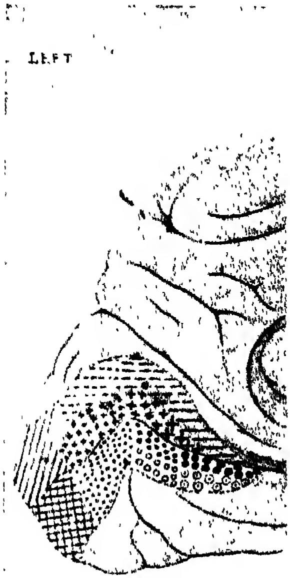


FIG. 62.—A diagram to illustrate the distribution of the cortical areas on the mesial surface of the occipital region of the left hemisphere. The two arrows indicate the plane of the section shown in Fig. 61. The area striata is represented by dots; the area parastriata by crosses; the area peristriata by circles. (After Elliot Smith.) Compare the above with Gordon Holmes's diagrams (Figs. 63, 64), arrived at on clinical grounds.

the upper retinal quadrant or lower field) running backwards in a more direct course to the upper part of the visual cortex (Fig. 219) They are close to the posterior cornu of the lateral ventricle, so that they are liable to pressure here when the ventricle is distended.

The occipital cortex in and about the calcarine fissure differs from the cortex elsewhere in the possession of a white line, the line of Gennari, interpolated in the grey matter. This area,

which is the primary visual or visuo-sensory area (Figs. 61–64), is the cortical projection of the corresponding halves of both retinæ. In this projection the part above the calcarine fissure



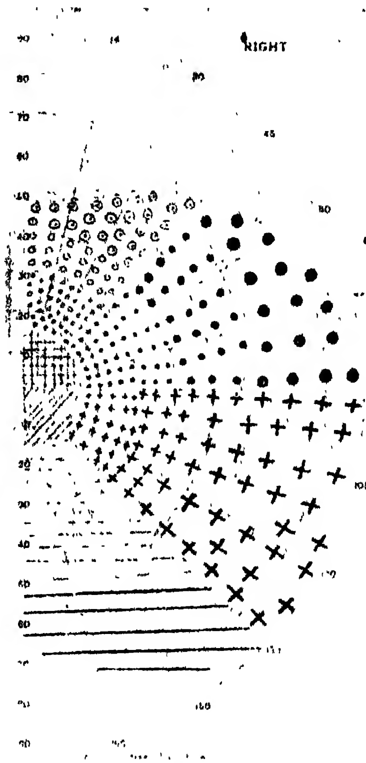


FIG. 63. FIG. 64.

FIG. 63.—A diagram of the probable representation of the different portions of the visual fields in the calcarine cortex. Fig. 63 is a drawing of the mesial surface of the left occipital lobe with the lips of the calcarine fissure separated so that its walls and floor are visible. The markings of the various portions of the visual cortex which is thus exposed correspond with those shown on the chart (Fig. 64) of the right half of the field of vision. (Gordon Holmes.)

represents the upper corresponding quadrants, the part below the lower corresponding quadrants of both retinæ, and the posterior part the macula.

SECTION II

THE EXAMINATION OF THE EYE

CHAPTER VI

External Examination

Ophthalmic patients may be roughly divided into two groups: those who present manifest objective signs of disease, and those who, presenting no outward and visible signs, have abnormal subjective symptoms. The division is convenient both in theory and in practice. The second group, in all but "functional" cases, have latent objective signs which it is our duty to discover. In the first group the manifest signs may mask even more serious conditions which it is our duty to bring to light. Not infrequently we are confronted with obscure cases which demand the most careful systematic examination in order that nothing may be overlooked. Though it is not always possible, or even necessary, to go through the lengthy routine of an exhaustive systematic examination, yet the details of such a routine must be firmly engrafted in our minds, ready for instant application, if we wish to avoid mistakes in diagnosis.

We shall first describe the methods of examination of the parts which can be observed with the unaided eye, though we shall often ensure accuracy by artificial assistance. Next, we shall describe the methods which must be employed to examine the deeper parts of the eye. Finally, we shall map out a routine of systematic examination.

In the patients who belong to our first group we shall at once be confronted with visible signs of disease. We shall not consider in this place gross signs such as marked protrusion or proptosis, great deviations of the optic axes from the normal parallelism, and so on. These will be more conveniently investigated in their special relations. We shall confine ourselves to conditions affecting the globe itself.

The Conjunctiva. In the normal position of the lids only that part of the bulbar conjunctiva which is exposed in the palpebral aperture, together with parts of the intermarginal strip along the edges of the lids, is visible. In order thoroughly to investigate the whole conjunctival sac it is necessary to expose the palpebral conjunctiva and the fornices.

The lower fornix is easily exposed by drawing down the lower lid while the patient looks towards the ceiling.

The upper palpebral conjunctiva is exposed by everting the upper lid.

Eversion of the upper lid requires some practice. (1) The best, and often the easiest, method is as follows: Stand facing the patient. Place the right index finger horizontally along the patient's left upper lid while he looks towards his feet. Draw the skin of the lid outwards: this causes the inner part of the edge of the lid to come forwards, while at the same time the pressure of the finger affords a fixed point around which the lid can revolve in a vertical direction. Insinuate the right thumb under the projecting edge of the lid, and roll the lid upwards towards the index finger. The right lid is everted in the same manner, using the left hand.

This method is very easy when the eyes are prominent, and it causes a minimum of discomfort to the patient. When the eyes are deeply set in the orbit, as is often the case in old people whose orbital fat has become to a great extent absorbed, more pressure is needed and a little pain is caused. In such cases the following method may be adopted; the tyro will generally find it easier:

(2) Place a probe or thin pencil horizontally along the skin of the upper lid at the level of the upper border of the tarsus, the patient looking towards his feet. Seize the eyelashes between the left index and thumb, and draw the lid away from the globe, using the probe as a fixed point. Rotate the lid in a vertical direction round the probe, which is then withdrawn.

In many cases we wish to evert the upper lid when standing behind the patient, who may be lying on a couch. In this case the following is the best method:

(3) Place the left index finger vertically upon the lid while the patient is looking towards his feet. Seize the lashes with the right index and thumb, and rotate the lid around the tip of the left index.

In babies a special arrangement of the patient facilitates thorough examination of the conjunctival sac and eye:

(4) The surgeon sits facing a nurse, who holds the child on her lap. The baby's head is placed between the surgeon's knees; its body is on the nurse's lap. She holds the child's hands against its body, thus keeping them out of the way, and at the same time steadying the child. If, as is often the case, there is blepharospasm, eversion of the lids is extremely easy; indeed, it becomes troublesome when we wish to examine the cornea. Here the spasm of the orbicularis fixes the lids against the globe, and the slightest attempt to draw the lids apart causes both to become everted. When this does not occur, method (3) must be adopted.

Having everted the upper lid we can examine the palpebral conjunctiva, but we are still unable to see the upper fornix. This can usually be effected in adults by the following manœuvre: (1) With the lid still everted by the first method it is fixed in that position by the left thumb placed upon its margin at about the middle. The right thumb or finger is placed in the middle of the lower lid. Firm, steady pressure is then made through the lower lid upon the globe in a direction straight backwards, as if to push the globe into the orbit. In the meantime firm pressure is also exerted backwards upon the upper lid with the left thumb. The fornix will generally start forwards suddenly, but only if the patient keeps looking well down towards his feet all the time.

This method, though unpleasant, is not painful. The only other method of exploring the upper fornix is more effectual, but also painful. The eye should therefore be well cocained.

(2) The upper lid is everted in the usual manner. A retractor is then inserted under the everted lid into the fornix. The margin of the lid being fixed as in (1), the lid is everted a second time, so that the fornix is fully exposed. Sometimes it is necessary to grasp the everted lid with forceps and thus evert it a second time.

By these manœuvres the conjunctival sac can be thoroughly explored. Special attention must be paid to the favourite sites for foreign bodies and manifestations of disease, *e.g.*, foreign bodies often lodge on the palpebral conjunctiva about 2 mm. from the margin at about the middle of the lid; trachoma follicles are most marked in the upper fornix; scarring from old trachoma is most marked in the palpebral conjunctiva, &c. Adhesions between palpebral and ocular conjunctiva and obliteration of either fornix cannot fail to be noticed. Inflammatory conditions can be thoroughly examined; accurate

diagnosis often depends upon minute investigation. Patches of granulation tissue which bleed easily may mark the site of embedded foreign bodies, or, if on the palpebral conjunctiva, the site of a chalazion.

The ocular conjunctiva can be almost completely examined without everting the lids if the eye is moved up and down while the lids are kept apart. The redness which is observed in irritative and inflammatory conditions varies in its distribution and nature according to the cause. Here we must remember the groups of vessels with which we have to deal (*vide* p. 11 ; Plate II.). Three groups may be distinguished, though most of them are too small to be recognised in health :

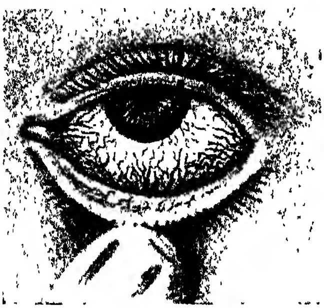


FIG. 65.—Conjunctiva congestion—engorgement of the posterior conjunctival arteries and veins. (After Guthrie.)

FIG. 66.—Congestion of the perforating branches of the anterior ciliary arteries. (After Dalrymple.) The dusky spots at the seats of perforation are often seen in dark-complexioned persons.

(1) the proper vessels of the conjunctiva or posterior conjunctival vessels ; (2) the anterior conjunctival vessels, which supply the area adjacent to the limbus or corneal margin and send minute loops into the edge of the cornea itself (Fig. 65) ; (3) the anterior ciliary vessels, lying in the subconjunctival or episcleral tissue (Fig. 66). In the last group the perforating branches of the arteries are seen in health as several comparatively large tortuous vessels which suddenly cease about 4 or 5 mm. from the corneal margin. They have very numerous small episcleral branches which are invisible in health, but when dilated form a pink zone of fine, straight, very closely set vessels around the cornea. The corresponding perforating veins are very small, but more numerous than

the arteries; their episcleral branches form a closely-meshed network

Congestion of the individual groups of vessels affords important evidence as to the seat of the mischief. The conjunctival vessels can be distinguished from the anterior ciliary by the following points: (1) they are a brighter brick-red, the ciliary vessels being seen through the conjunctiva, which imparts a purple tinge; (2) if the conjunctiva is moved to and fro over the sclerotic by the finger placed on the lower lid, the conjunctival vessels also move while the ciliary remain stationary; (3) the individual vessels and the network they form can be seen in the conjunctival system, whereas the ciliary form for the most part a diffuse reddish-violet blush in which the separate vessels are indistinguishable; (4) if the blood is

FIG. 67.—Ciliary congestion—engorgement of episcleral twigs of the anterior ciliary arteries. (After Dalrymple.)

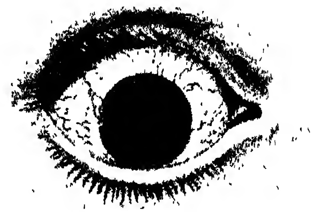


FIG. 68.—Congestion of the anterior ciliary veins, episcleral venous plexus. (After Dalrymple.)

pressed out of the vessels the ciliary fill up at once on removing the pressure, since they anastomose very freely, while the conjunctival fill slowly.

In general, congestion of the conjunctival vessels, leaving a relatively white zone around the cornea, accompanied by mucous or muco-purulent secretion, is indicative of conjunctivitis. If there is much irritation and so-called photophobia, with some blepharospasm and very watery—lacrymal—secretion, we suspect the presence of a foreign body on the cornea or under the lid, so that it rubs against the cornea: the condition may be due to misplaced lashes (trichiasis). Phlyctenular ophthalmia may produce a similar picture. In such a case there is also usually congestion of the anterior conjunctival vessels. Careful examination shows that the vessels in the circumcorneal zone are bright red, and that the corneal

loops are also dilated and visible. Any irritation of the cornea—ulcers, abrasions, &c.—causes this conjunctival congestion of the circumcorneal zone and corneal vessels. Though conjunctival they do not move with the membrane. A definite leash of dilated vessels confined to the conjunctiva or encroaching upon the cornea is usually indicative of a foreign body on the cornea or phlyctenular disease.

Pink circumcorneal congestion is also met with in inflammation of the iris. Here the anterior ciliary vessels are also involved.

Circumcorneal congestion of a peculiar lilac tint, more deeply seated and often patchy, is associated with cyclitis and deep scleritis. This is the condition which is known as ciliary congestion *par excellence.*

Dusky congestion at the limbus, composed of a fine reticulum—the episcleral venous plexus—often points to glaucoma, but may accompany other diseases, especially in old people.

These conditions run into one another very frequently, so that they then cease to have special diagnostic importance.

Lacrymal Apparatus. Conjunctival congestion of one eye only, or signs of irritation such as watering, should lead us to suspect the efficiency of the lacrymal apparatus. Simple epiphora or flow of tears on to the cheek may be due to blocking of one or both puncta or to their malposition, or to blockage elsewhere in the canaliculi or nasal duct. Displacement of the lower punctum may be easily overlooked. The puncta are not visible normally without slightly everting the lids. Displacement is often very slight, due to slackness of the lid causing a little rolling out or ectropion, especially in old people. In spasm of the orbicularis the lid may be rolled in too much (entropion); this may also cause epiphora. Sometimes with more conjunctival inflammation, but often without, there is distension and chronic inflammation of the lacrymal sac. In all such cases we carefully observe the exact position of the puncta, whether they are in apposition to the bulbar conjunctiva as they should be, and also whether there is any regurgitation from the lacrymal sac when it is pressed upon. The lacrymal sac is situated in the lacrymal fossa between the inner canthus and the nose: the fundus of the sac extends slightly above the level of the inner palpebral ligament, which is on a line with the canthus. Pressure inwards and backwards in this position will press upon the sac. If there is any obstruction to the flow of the contents into the nose by the nasal duct, as is usually the case when the sac is inflamed,

the contents will generally regurgitate into the conjunctival sac by way of the canaliculi, and will be seen pouring from the puncta. We note whether the contents are tears, mucus, or muco-pus.

Without describing special methods of ascertaining if the lacrymal passages are patent (Chap. XXXII.), a simple test may be mentioned. A drop of fluorescein solution is placed in the conjunctival sac and the patient is told to blow his nose. If the passages are free, the handkerchief will be stained with the solution.

The Sclerotic. Inspection of the sclerotic around the cornea may reveal raised nodules of episcleritis with their localised areas of vascular congestion (*vide* p. 251). Deep scleritis may be shown by dusky ciliary congestion and opacification of the deeper layers of the cornea at the periphery (sclerosing keratitis, *vide* p. 253).

Definite blue coloration of the circumcorneal sclerotic, except in young children, is pathological. It is most frequently due to ciliary staphyloma, a giving way of the sclera owing to inherent weakness (injury, scleritis, &c.) or to increased intraocular pressure (glaucoma). Discoloration may be due to pigmentation. Slight duskiness around the spots where the anterior ciliary vessels perforate is not uncommon in people with dark complexions. Otherwise pigmentation in this neighbourhood, either in the conjunctiva or sclerotic, should be regarded with suspicion as indicative of melanotic sarcoma. Definite nodules of deeply pigmented tissue in the situation of the perforating vessels are very significant of sarcoma of the ciliary body.

If there is bulging of the sclerotic an attempt should be made to transilluminate it in the dark room. If it is a true ectasia (staphyloma) light will pass through into the eye. The light should be concentrated upon the spot by a strong lens (*vide* p. 87).

The abrupt or very gradual curvature of the sclerotic as it passes back from the cornea may indicate high hypermetropia or myopia respectively.

The Cornea. A little experience will enable us to recognise at a glance if the cornea is smaller than usual. A small cornea with a shallow anterior chamber is very suggestive of glaucoma.

The cornea should be bright and transparent. We first examine its surface. Any loss of substance, such as an abrasion, may easily be overlooked without special care.

The condition of irritation and lacrymation will often put us on the track, but these features may be so slight as to pass unnoticed. The following methods should be adopted :

(1) Place the patient facing the window. Stand in front and direct the patient to follow the index finger, which is held horizontal and moved slowly up and down. The finger is then

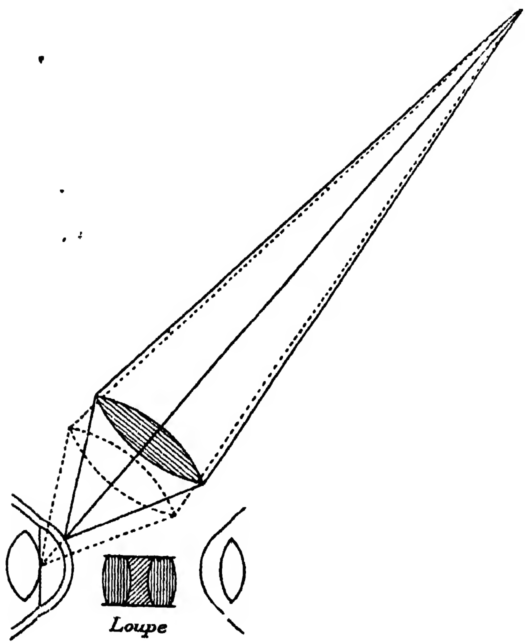


FIG. 69.—Focal or oblique illumination. The diagram shows how the focus of the light may be altered by slightly shifting the position of the concentrating lens.

held vertical and moved from side to side. While these manœuvres are being carried out the image of the window, or corneal reflex as it is called, is carefully watched. If the surface of the cornea is normal there will be no distortion of the reflex as it passes over it. If there is an abrasion the image will be distorted here, and will be less clearly defined. This method should always be resorted to first, as it is good practice in observation and needs no artificial aid, which may not be available in some circumstances.

(2) If the first method gives uncertain results or fails, instil a drop of fluorescein solution (2 per cent.). This is best done by telling the patient to look towards his feet; the conjunctiva above the cornea is then lightly touched with the tip of a glass rod which has been dipped in the solution. A pad of cotton wool is immediately placed upon the closed lids so as to mop up the excess and the tears which tend to flow over the face. If this detail is not attended to the face becomes stained unnecessarily. It is a good plan to wash out the excess of fluorescein with a drop of pantocain solution, but it is not essential. Any spot on the surface of the cornea which is denuded of epithelium will appear green.

A bundle of dilated conjunctival vessels near the limbus will often point to the site of a foreign body upon the cornea.

Opacities of the cornea may be so faint that they require very minute investigation, and the same is true of the details —depth, &c.—of gross opacities. We can study them best by focal or oblique illumination.

Focal or oblique illumination is carried out as follows (Fig. 69): the patient is placed, preferably in the dark room, with a light about two feet in front but slightly to one side. The light is concentrated upon the cornea by a strong convex lens. The rays of light are brought to a focus by the lens. The cornea or other superficial structures can thus be examined under the intense light of the converging rays. The position of the minute image of the light formed by the lens can be moved over the surface of the cornea by slight lateral movements of the lens without altering the position of the light. Similarly the light may be focussed upon the iris or crystalline lens by moving the lens slightly nearer to the eye. A small electric torch is a convenient source of illumination.

Having thus brilliantly illuminated the part of the cornea which we wish to investigate, we may magnify the spot by looking through a very strong convex lens or corneal loupe held in the other hand. The management of the two lenses requires a little practice, but is easily mastered. A few words of explanation about the corneal loupe will help us to employ it to best advantage.

When we magnify a small object with a strong convex lens we place it within the focal distance of the lens and view it through the lens. We know that under these conditions the lens forms an enlarged image upon the same side as the object, but farther away (*vide* p. 30, Fig. 21). In order that we may see the image to best advantage we must see as much of it as

possible, and we must see it under the largest possible visual angle. The first requisite demands that the observer's eye shall be as close to the lens as possible. The second requisite depends upon the relative distances of the object and the eye from the lens; in practice these are found by slight movements of the lens.

In employing focal illumination, then, first focus the light upon the required spot. Then place the corneal loupe near the spot and look through it. Slowly advance the loupe towards the cornea until the spot comes into focus. Then get one's eye as close to the loupe as possible.

By moving the light and the loupe slowly over the whole surface of the cornea we can thoroughly explore it. By advancing the convex lens we can illuminate successively the back of the cornea, the iris and anterior part of the lens, and finally the deeper parts of the lens. By simultaneously advancing the position of the loupe towards the cornea we can successively bring these structures into accurate focus and examine them under considerable magnification. We cannot get beyond the back of the lens with a high power loupe, as it works at too short a focal distance. Moreover, in order to examine the deeper parts of the lens, we must have the light almost in front of the patient, otherwise they will not be illuminated.

With a binocular loupe a stereoscopic effect is obtained, and the depth of opacities can be determined with great accuracy, but the degree of magnification is less. Special methods of examination with the binocular loupe, Gullstrand's slit lamp, contact illumination, &c., are useful in difficult cases (*vide* p. 97).

Focal illumination without the assistance of a loupe or a dark room is often of great advantage through the good illumination which it affords. One soon gets into the habit of concentrating the light from the window upon the eye with the convex lens with a view to improving the optical conditions.

The cornea is often affected secondarily to the conjunctiva, as in phlyctenular ophthalmia. In such conditions the eye is most irritable and resistant to examination in bright light. The slightest attempt to separate the lids is accompanied by violent blepharospasm, especially in children. Yet it is in such cases that it is of the greatest importance to know and watch the condition of the cornea. For aught we know it may be ulcerated, and may even be upon the point of becom-

ing perforated. Any roughness, or even an amount of pressure which is quite justifiable in other cases, may suffice to cause the perforation which it should be our chief effort to avoid. In such a case the lids must be separated by retractors. We may use simple bent wire retractors (Fig. 70) or Desmarres' retractors (Fig. 71). In babies the position recommended in method (4), p. 81, is employed: older children are placed upon a couch. The retractors are inserted gently into the palpebral aperture, first the upper, then the lower, the curved ends being insinuated between the lids and the globe. Traction is then made upon the retractors, not only in opposite directions up and down, but also away from the globe, so that the lids are lifted off the globe at the same time that they are separated. In these cases the globe tends to roll forcibly upwards when light falls upon it, which makes it very difficult to see the cornea even with the use of retractors. The difficulty may be overcome by pressing the end of the lower retractor well into the lower fornix, which drags the eye downwards by pulling on the ocular conjunctiva.

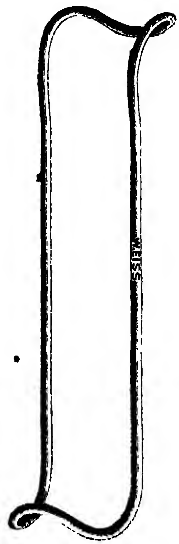


FIG. 70. — Bent wire lid retractor.

When we have satisfied ourselves that there is little or no fear of perforation we may dispense with retractors. It has been pointed out that in children an attempt to separate the lids when there is much blepharospasm is usually followed by eversion of both lids, the cornea still remaining hidden. The way to overcome this difficulty is to place the two thumbs close to the edges of the lids and to press gently but firmly upon the globe as the lids are drawn apart. In this manner they are separated without becoming everted, but we must be extremely careful not to exert undue pressure and not to touch the cornea with the thumb nails.

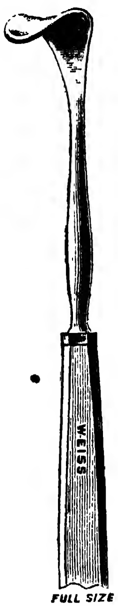


FIG. 71. — Desmarres' lid retractor.

In many diseases new vessels are formed in the cornea. An

exact knowledge of their position, whether superficial or deep, and of their distribution, whether localised, general, peripheral, above, and so on, will often settle a disputed point in diagnosis.

Superficial vessels (Fig. 72) in the cornea are distinguished

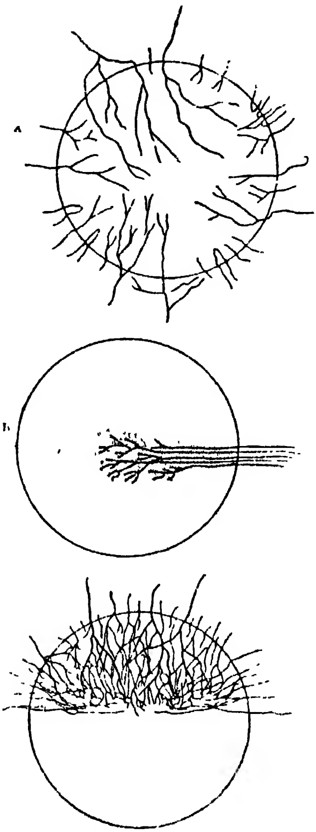


FIG. 72.—Diagrams of superficial corneal vessels. A, general vascularisation; B, a single leash of vessels, as in fascicular ulcer (*vide* p. 221); C, localised vascularisation, as in trachomatous pannus (*vide* p. 178).

from deep (Fig. 73) by the following features: (1) superficial vessels can be traced over the limbus into the conjunctiva, while deep ones seem to come to an abrupt end at the limbus; (2) superficial vessels are bright red and well defined, while deep ones are ill defined, greyish red, or cause only a diffuse

red blush; (3) superficial vessels branch in an arborescent fashion, dichotomously, while deep ones run more or less parallel to each other in a general radial direction, and branch at very acute angles, like a besom; (4) superficial vessels may raise the epithelium over them so that the surface of the cornea is uneven, while with deep ones the cornea, though hazy, is smooth. The peculiar course of deep vessels is probably determined by the lamellar structure of the substantia propia.

The sensibility of the cornea may be tested by touching it in various spots with a wisp of cotton-wool twisted to a fine point and comparing the effect with the opposite side. Normally there is a brisk reflex closure of the lids. The sensibility is often diminished in corneal affections, but the change is of some diagnostic significance in certain cases, *e.g.*, herpes (*vide* p. 229).

The Anterior Chamber. The anterior chamber is shallow in extreme youth and in old age: at other periods of life it is about 2·5 mm. deep normally. It must be remembered that we estimate the depth of the anterior chamber by the position of the iris, and that we view the iris through the cornea, which is a strongly refracting convex surface. The effect of this is to magnify the iris and pupil, and to make it appear farther forwards than it really is. The same applies to anything in the anterior chamber, *e.g.*, the point of a knife in operations.

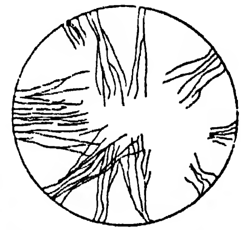


FIG. 73. — Diagram of deep corneal vessels, as in interstitial keratitis (*vide* p. 235).

Good binocular vision enables us to estimate the depth of the anterior chamber when we are looking at it from in front. The observation should be confirmed by taking a profile view.

The anterior chamber is abnormally shallow in glaucoma. It is often abnormally deep in irido-cyclitis. It is frequently unequal in depth in different parts. For example, it may be deeper at the periphery than in the centre in irido-cyclitis; on the other hand, when the iris is bowed forwards (iris bombé) it is funnel-shaped, the centre being deep, the periphery very shallow. It may be deeper on one side than on the other owing to tilting (subluxation) of the lens.

After considering the depth, attention must be paid to the contents. In some wounds and ulcers of the cornea, and rarely without them, there is pus in the anterior chamber. It

forms a layer at the bottom, the surface of the pus being level (hypopyon). A similar layer of blood may occur after contusions or spontaneously (hyphæma). The aqueous may be hazy, a condition not always easy to distinguish from haziness of the cornea. Such cases lead us to examine very carefully the back of the cornea with the loupe under focal illumination to see if there are any precipitates ("keratitis punctata") upon it, or we may see flocculent specks in the aqueous. All these conditions are of great diagnostic and prognostic importance.

The Iris. We pay attention first to the *colour* of the iris and the clearness of its pattern. The two irides or parts of the same iris may be of different colour, both conditions being known as heterochromia iridis. A grey iris, with ill-defined pattern, suggests atrophy from cyclitis, glaucoma, &c. Dark brown spots in the iris, not raised above the surface, are common. Care must be taken to distinguish them from small nodules of the same colour or white (sarcoma, tubercle, gumma). "Muddiness of the iris" is the expression used for indistinctness of the pattern, caused by inflammatory exudates. A muddy iris, with small irregular pupil and sluggish reaction to light, is indicative of iritis.

The *position* of the iris must be noted, especially the plane in which it lies (*vide* pp. 263, 272, 285). Special attention should be paid to any adhesions (synechiæ), anterior—to the cornea, or posterior—to the lens capsule. *Tremulousness* of the iris (iridodonesis) is seen when the eyes are moved rapidly if the iris is not properly supported by the lens, *e.g.*, in absence, shrinkage, or dislocation of the lens, slackness of the suspensory ligament, &c. It is best seen in a dark room with oblique illumination.

The Pupils. A point which should be examined at an early stage in every routine examination of the eyes is the condition of the pupils. This is the more important since the routine examination frequently demands the use of a mydriatic, and if the pupils have not previously been noted it may be necessary to require the attendance of the patient on another occasion when the effect of the mydriatic has passed off.

The examination of the pupils requires careful attention to details if trustworthy results are to be obtained. It is best carried out as follows:

Place the patient facing the light, which should not be too bright; see that the two pupils are equally illuminated. Note the size, shape and contour of each pupil. Cover both eyes

with the palms of the hands, preferably without touching the face. Tell the patient to look straight at you. Remove one hand and watch the pupil. Replace this hand and remove the other, watching the other pupil. Note down and compare the results (*direct reaction to light*). Remove one hand so that this eye is exposed to light (it should be shaded from intense light). Watch this pupil as the hand is removed from the other eye. Repeat the process whilst watching the other pupil (*consensual reaction to light*).

Now tell the patient to look quite across the room, as far off as possible. Suddenly hold up the index finger vertically, at about six inches from the patient's nose, and tell the patient to look at it. Watch the pupils while he accommodates for the finger (*reaction to accommodation*).

When the reaction to light is feeble and the pupils are already small, it is difficult to be certain of the results in bright diffuse daylight, the corneal reflexes adding to the difficulty. In such cases the patient should be taken into the dark room and the light concentrated upon one pupil by focal illumination. By a slight lateral movement of the convex lens (*vide* p. 87) the focus of light can be moved on or off the pupil, the pupillary movements being watched the while. If there is no movement under these conditions we may conclude that the reaction to light is absent.

It is better not to use the ophthalmoscope mirror in this procedure, as the patient is very likely to look at it, and a reaction to accommodation may be mistaken for one to light. Note very carefully if the constriction of the pupil to light is well maintained (*vide* p. 395).

The same method will elicit the *hemianopic pupil reaction* (Wernicke) in the rare cases (lesion of one optic tract) in which it is present. To test for it, the light is placed in front, but rather to one side of the patient. The light is focussed with the lens upon the opposite side of the retina, and the pupil watched. The light is then moved to the other side and is now focussed on the other side of the retina. The best source of illumination for this purpose is the diffuse light from a large window (Fisher). If the reaction is present the pupil will react briskly when one half of the retina is illuminated, but very slightly or not at all when the other half is illuminated. It usually reacts slightly even in the latter case, owing to the impossibility of preventing diffusion of light on to the sensitive half of the retina, and for this reason the test is rarely unequivocal.

When the pupils are small to start with ("spinal miosis"), do not react to light, but react to accommodation, the condition is known as the *Argyll Robertson pupil* (*vide* p. 595). It occurs especially in para-syphilitic disease, most commonly and in its most characteristic form in tabes, and frequently in general paralysis of the insane; but it is also found in other syphilitic diseases of the central nervous system.

If the above directions are carried out we shall have reliable information as to the shape and relative size of the pupils and their reactions. A few of the commoner conditions may be enumerated here.

Very large pupils will suggest that a mydriatic has been used. It is not uncommon for it to have been used inadvertently. We not infrequently see a patient with the right pupil widely dilated complaining of dimness of vision. Inquiry will often elicit the fact that he has been using a liniment for rheumatism. The explanation is that the liniment contained belladonna, and that after using it with his right hand he rubbed his right eye with the soiled fingers. Often patients use ointment or drops prescribed for other patients. We must always be on our guard against such traps. These pupils are usually quite immobile, and the patient complains of dimness of vision, especially in near work.

The pupil is also large and immobile in complete atrophy of the optic nerve; this may be due to absolute glaucoma. In acute glaucoma it is usually large, immobile, and oval, with the long axis vertical; the condition is generally unilateral. If only one eye is blind from disease of the optic nerve this pupil is rather larger than its fellow as a rule, but the consensual reaction to light on the sound eye appears to be much increased, granted of course that the third nerve is intact. Dilatation of the pupils with retained mobility is found sometimes in myopia and in conditions of impaired nerve tone, *e.g.*, anæmia; it is also found in cases of disseminated sclerosis with optic atrophy, which rarely if ever leads to complete loss of sight in this disease. Dilatation as a reflex to painful impressions has already been mentioned. Unilateral dilatation may result from irritation of the cervical sympathetic in the presence of glands in the neck, pneumonia, phthisis, chronic pleurisy, cervical ribs, thoracic aneurysm, &c., but it is rare from such cause: it may also be due to syringomyelia, acute anterior poliomyelitis and meningitis affecting the lower cervical and upper dorsal part of the spinal cord and to pressure on the sympathetic fibres leaving the cord in the lower cervical and upper dorsal ventral

roots. Many of these causes lead eventually to constriction of the corresponding pupil from paralysis of the sympathetic. Temporary dilatation of one pupil is not very uncommon. Unilateral dilatation with immobility may result from a blow on the eye (Chap. XXI.).

The pupils are small in babies and in old people. When the pupils are small, as under the influence of bright light, the relation to the centre of the cornea can be best seen: the centre of the pupil is usually a little to the nasal side of the centre of the cornea. Small pupils are rarely perfectly round. A small immobile pupil should make us suspect old iritis with posterior synechiæ, and should lead to investigation with a mydriatic—homatropine for diagnostic purposes—to see if the pupil dilates regularly. Bilateral small immobile pupils make us suspect disease of the central nervous system (*e.g.*, pontine hæmorrhage); further examination may show that the immobility is confined to reaction to light (*vide* p. 94). A small sluggish pupil, with muddiness of the iris, is associated with iritis, which may be primary, or secondary to corneal trouble.

Very small immobile pupils suggest the use of drugs, either locally, *e.g.*, eserine, or through the general system, *e.g.*, morphia.

The chief causes of inequality of the pupils have already been mentioned incidentally. As indicative of central nervous disease it is found in general paralysis and tabes.

We must note carefully the nature of the contraction when the pupil reacts to light, and especially if the constriction is well maintained (*vide* p. 395).

Direct reaction to light does not eliminate the possibility of the patient being blind, *e.g.*, in uræmia and post-basic meningitis.

The Lens. The lens cannot be thoroughly examined without the assistance of the ophthalmoscope. By inspection, aided if necessary by focal illumination, we note any opacities in the pupillary area. The pupil may be blocked with iritic exudates (inflammatory pupillary membrane, blocked pupil).

Opacities in the lens itself are seen by oblique illumination as grey, white or yellowish patches. According to their distribution and nature we diagnose the various forms of cataract, but our observations must always be confirmed and controlled by ophthalmoscopic examination. The following example will show how easily one may be led astray. When the light is concentrated by focal illumination upon the pupil of a young person's eye the lens substance seems almost perfectly clear;

at most we see a faint bluish haze. If we examine the lens of an old person in the same manner the haze is much more pronounced; the lens substance in fact looks slightly milky. We might conclude that the patient has cataract. Examination with the ophthalmoscope will, however, show a perfectly clear red reflex. The explanation is that the lens substance generally becomes more optically dense, *i.e.*, the refractive index increases, as the person grows older (*vide* p. 53). Now the higher the refractive index of a substance the greater will be the scattering of light from its surface. The milkiness which we see is due to rays of light which are reflected from the lens and enter our eyes. The more rays reflected the more will the lens appear milky.

If, however, the white appearance is very pronounced, and especially if it is strictly localised to certain parts of the lens substance, we may safely diagnose cataract. A spot in the centre of the pupil, looking as if it were on the surface of the lens, may be a pupillary exudate or an anterior polar cataract. Triangular spokes of opacity with their apices towards the centre are indicative of senile cataract. A very white appearance over the whole pupillary area suggests a total cataract; if it is yellow and the iris is tremulous we suspect a shrunken calcareous lens.

The Tension. Last in the external examination, but by no means of least importance, we test the tension of the walls of the globe, which is increased when the intraocular pressure is raised, though not necessarily *pari passu*. It is done in the same manner as testing for fluctuation in other parts of the body.

Stand facing the patient, who is told to keep looking towards his feet. Place the index fingers of both hands side by side and touching each other upon the upper lid, steadying them by the other fingers lightly applied to the brow. Keep one finger quite still, pressing upon the globe through the lid. Now attempt gently to indent the globe with the other finger, pressing directly downwards, concentrating the attention meanwhile on the impression which is conveyed to the stationary finger. Repeat the process on the other eye.

The student should practise this manœuvre on a number of healthy eyes. He will thus obtain a mental estimate of what is to be considered normal tension—Tn. In absolute glaucoma the eye is usually stony hard; this condition is generally indicated by the convention T + 3, a misuse of numbers, but one in common use. The gradations of in-

creased tension from Tn to T + 3 are usually indicated by the conventions T full (T +), T + 1, T + 2. Similarly the gradations of diminished tension are represented by T minus (T —), T — 1, T — 2, T — 3.

Instruments known as *tonometers* have been devised for measuring the tension of the intact eye. They are far less reliable than the manometer, which, however, cannot be used on the human eye. The best tonometer for clinical use is that invented by Schiötz (Fig. 74). With it the depth of the indentation in the cornea, anæsthetised with 1 per cent. pantocain solution, made by a weighted stylet is measured by a lever which travels over a scale. There are four weights (5·5, 7·5, 10 and 15 gms.) and the greatest accuracy is attained with the weight which gives a deflection of the lever of 2 to 4 mm. The instrument is calibrated so that the equivalents of the readings in millimetres of mercury can be read off a chart. The readings are inaccurate when transformed into pressures in millimetres of mercury, but the tonometer is certainly useful for comparative measurements, *e.g.*, between the two eyes or between successive measurements on the same eye.

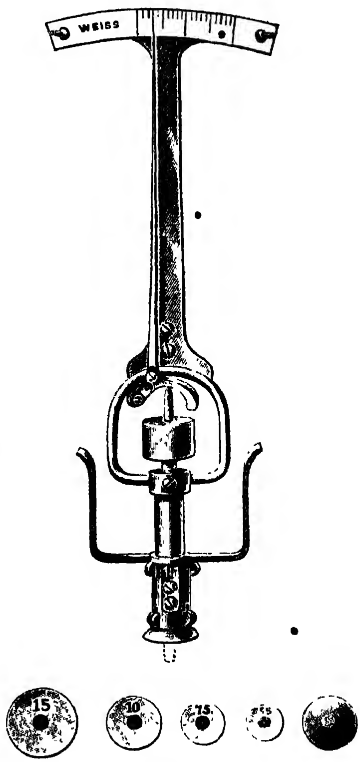


FIG. 74.

The Microscopy of the Living Eye. The invention of the slit-lamp by Gullstrand has rendered possible the examination of the anterior parts of the eye under considerable magnification by the binocular microscope. The somewhat complicated technique militates against its routine use as a clinical instrument, but it has already added to our knowledge of anatomical and pathological conditions in the cornea, anterior chamber, iris, lens and vitreous. The most important results are referred to elsewhere under the appropriate headings. Fig. 75 shows a

general view of the eye illuminated by a beam of light of moderate width coming from the slit-lamp entering the eye from the left side. Optically homogeneous media appear quite black; structures like the cornea, lens, and suspended particles in the aqueous scatter the light. Hence, on the left of the diagram is seen the illuminated portion of the cornea forming a parallelepiped, the brighter area corresponding to the surface, the darker to the

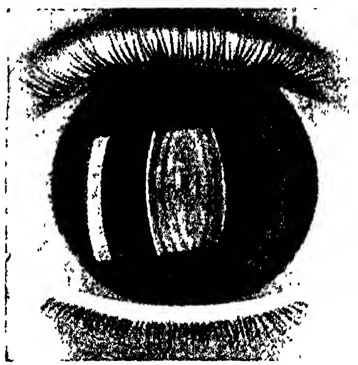


FIG. 75.—Optical section of the eye. The zones of optical discontinuity of the cornea and lens are clearly seen, and the aqueous flare is indicated as well as the structure of the vitreous. The drawing, of course, is composite as if all parts of the beam were in focus simultaneously.

section of the cornea. The black space to the right is the anterior chamber. Then follows the "phantom" of the lens, in which can be distinguished the dim central interval, the **Y** and **⅄** which delimit the fœtal nucleus anteriorly and posteriorly, and the surfaces of the adult nucleus (*vide* p. 9). Still farther to the right is the faintly striated vitreous.

Since minute details, *e.g.*, particles floating in the aqueous, are revealed by the slit-lamp, considerable experience in its use is necessary in estimating their pathological significance.

CHAPTER VII

Ophthalmoscopic Examination

THE internal parts of the eye beyond the lens cannot be seen without the assistance of the ophthalmoscope. A little consideration of the optical conditions of the eye will show the reason.

Under ordinary circumstances the pupil looks black, and no red reflex, much less a clear image, is obtained from the fundus. If, as in Fig. 76, there is a source of light, L, in front of the eye, and the eye is focussed upon it or accommodated for it, the light and a spot upon the retina are conjugate foci; *i.e.*, the image of the spot of light is a spot on the retina.

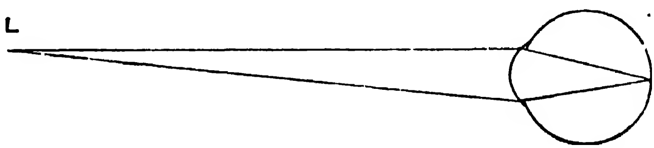


FIG. 76.

Reversing the direction of the rays, all rays from the illuminated spot of the retina are brought to a focus at the source of light. Therefore no rays will enter an observing eye unless it is situated actually at the source of light. The problem solved by Babbage (1848) and rediscovered by von Helmholtz when he invented the ophthalmoscope (1851) was practically that of making the observing eye at the same time the source of illumination of the observed fundus.

If the eye is not focussed for the source of light the conditions are different, and some slight luminosity of the pupil may be seen. This is one cause of luminosity in the pupils of the hypermetropic eyes of young children and most carnivora. Extreme hypermetropia is also the cause of the so-called amaurotic cat's eye, which is due to detachment of the retina, glioma of the retina, &c. In these cases the retina is pushed forwards and the fundus at this spot becomes highly hypermetropic, the reflex from the pupil being often the first symptom noticed. The same principle applies to the reflex

from the eye after the lens has been removed by extraction of cataract.

In hypermetropia the conjugate focus of the source of light, L, is a point, l, behind the retina (Fig. 77). Hence the emergent rays from the illuminated area of the fundus are divergent, as if coming from l. Therefore an observing eye situated anywhere within the area l_1 l_2 of the cone of emergent rays will catch some of them, and the pupil of the observed eye will appear feebly illuminated. Under these circumstances it is not necessary for the observing eye to occupy the exact position of the source of light, but only a spot in its immediate neighbourhood. On the same principle, the extremely hypermetropic retina in glioma retinæ, &c., can be seen well by focal illumination.

In high myopia the emergent rays are strongly convergent,

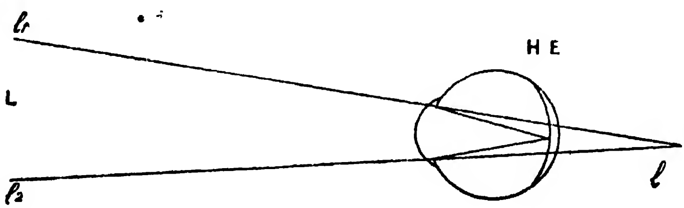


FIG 77.

and become divergent after coming to a focus at the remote point (Fig. 80). Beyond this point some of the divergent rays may enter an observing eye suitably situated and the observed pupil appears illuminated.

The luminosity of albinos' eyes is due to light entering the eye, not only through the pupil, but also through the iris and sclerotic. That this is the true explanation is shown by the fact that the pupil looks black if it is observed through a small hole in an opaque screen. A small amount of light passes through the sclerotic in the normal eye.

It will help us to understand the principles of the ophthalmoscope if we say a few words about its historical development. The ophthalmoscope was invented by Babbage in 1848, but its importance was not recognised, and it was rediscovered by von Helmholtz in 1851. The original ophthalmoscope of von Helmholtz was merely a plane plate of glass (Fig. 78). A source of light was placed beside the observed eye and the glass plate obliquely in front of it, so that a portion of the light was reflected from the surface of the plate into the

eye. On looking through the transparent plate an observer could now receive some of the rays from the fundus into his own eye, and thus obtain an image of the illuminated fundus. Since but a small proportion of the light received upon the plate is reflected at its surface the illumination is feeble. Nevertheless, the principle is worth bearing in mind as a ready means of getting a view of a fundus in the absence of a more satisfactory ophthalmoscope. Moreover, an error of refraction in the observed eye may be obviated by using the corre-

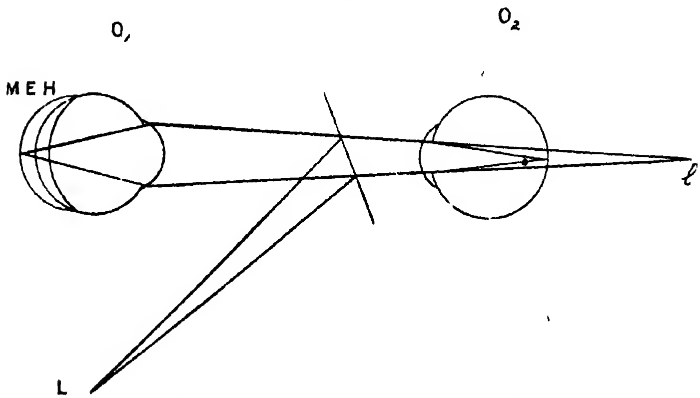


FIG. 78.—Diagram of von Helmholtz' ophthalmoscope. O_1, observed eye; O_2, observer's eye; L, source of light; *l*, image of L formed by the plane mirror—immediate source of light; M E H, relative positions of retina in myopia, emmetropia, and hypermetropia respectively, showing the relative sizes of the areas of retina illuminated in each case.

sponding spectacle glass of the patient as the ophthalmoscopic mirror.

Von Helmholtz next increased the amount of light reflected by superposing three plane plates. The back of the glass was next converted into a more powerful mirror by silvering it, leaving a small portion unsilvered or leaving a hole in the mirror, through which the observer might look. The illumination was still feeble, since the rays reflected by a plane mirror are divergent (*vide* p. 25). Ruete therefore (1852) introduced the perforated concave mirror which still holds the field. The final modification was the addition of a battery of small lenses of various strengths, which might be brought into position behind the aperture. The multitudinous forms of

"refraction ophthalmoscopes" are merely various mechanical contrivances for doing this most conveniently.*

There are two chief methods of ophthalmoscopic examination, the direct method (v. Helmholtz, 1851) and the indirect method (Ruete, 1852). The ophthalmoscope is provided with two mirrors, one a small one, slightly tilted, for the direct method, the other a large one, not tilted, for the indirect method. Both are concave, the former with a focal distance of 30 cm., the latter of 10 cm. It is an advantage to have also two plane mirrors corresponding with these; with such a four-mirror ophthalmoscope the surgeon is fully equipped for every detail of ophthalmoscopy and retinoscopy.

The importance of system in his methods is so often impressed upon the student that he is liable to underrate it through sheer reiteration. It will perhaps suffice to say here that in using the ophthalmoscope he will inevitably come to grief unless he pursues his examination on a well-ordered plan. The order of examination should be as follows:

(1) Preliminary examination with the mirror alone at a distance of about 1 metre from the patient;

(2) Examination with the mirror alone at a distance of about 20 cm. (reading distance) from the patient; this is sometimes called the distant direct method;

(3) Examination by the indirect method;

(4) Examination by the direct method.

The following facts will impress upon the student the reasonableness of this procedure. By (1) we obtain knowledge of the nature of the refraction of the eye under examination; this will prevent many little difficulties when we come to closer quarters. By (2) we see any gross changes, especially opacities in the refractive media; these may be made at once evident by this method, whereas they may be very puzzling

* The student is advised to procure a good ophthalmoscope at the outset of his clinical work in the medical wards. The cheaper forms are not only waste of money, but are a perpetual source of annoyance. The modification of Couper's ophthalmoscope, generally known as Morton's, is most strongly recommended. In recent years various self-luminous ophthalmoscopes have been devised. In most of these half the aperture of the ophthalmoscope is used for illuminating the fundus from a small electric bulb contained in the instrument, the other half being used for observation. The lamp is run off a small dry battery which may be placed in the handle of the ophthalmoscope. In the polarising ophthalmoscope the light is plane-polarised, thus eliminating the troublesome corneal reflex (*vide* p. 109). Self-luminous ophthalmoscopes are particularly useful for examining bed-ridden patients. They have the disadvantage that they are seldom suitable for examination by the indirect method—*a method which should never be omitted before using the direct method.*

if first observed by (3) or (4). In addition, we shall see the details of any very hypermetropic part of the fundus, such as a detached retina or glioma of the retina; these also are by no means difficult to miss by (3) and (4). By (3) we get a general view of the fundus—the largest possible area under moderate magnification; it is exactly comparable to microscopic examination with a low power. By (4) we examine details under a higher magnification; it is exactly comparable to microscopic examination with a high power.

The student should begin by taking a patient whose pupils have been dilated with atropine, *e.g.*, a boy of twelve or fourteen who has come for the correction of his refraction. The atropine will have had the additional advantage of having paralysed the patient's accommodation. The observer should know his own refraction.

The patient is taken into the dark room and seated beside the light. The light is placed to the side which is to be examined, but well behind the level of the patient's face; the eye should be as much as possible in darkness. The observer sits facing the patient, about a metre from him. He reflects the light from the large ophthalmoscope mirror into the eye, meanwhile looking through the sight-hole. This requires a little practice, but is quickly mastered. When the light falls on the eye he notices a red reflex from the pupil. There ought to be no black spots in the pupillary area, but either a uniform red reflex or obscure details of the fundus. By tilting the mirror to and fro in various directions he can obtain an approximate idea of the refraction of the eye (*vide* p. 107).

The observer now stands up and approaches the patient until his eye, still with the large mirror, is about 9 inches from the eye under observation. He can now see the cornea and iris clearly, and can confirm any points which he has made out previously by the external examination.

He then sits down again at about a metre from the patient. Still keeping the light upon the eye with the large mirror, with his left hand he holds the large convex lens, which he will find in the ophthalmoscope case, close in front of the eye. He will be wise not to hold the lens absolutely vertical, but to tilt it very slightly. He will probably see only the magnified iris through the lens. He now watches the red reflex from the pupil, and slowly withdraws the lens from the eye towards himself. At a certain point he will see an inverted image of the fundus quite clearly. The indirect method requires also some practice, but the amount required may be

much diminished and much greater accuracy attained if the optical conditions under which the examination is made are thoroughly understood. These will be explained immediately.

Having obtained a good general view of the fundus, the observer again approaches the patient. He now uses the small tilted mirror of the ophthalmoscope. The mirror is tilted so that it faces towards the light. Looking through the sight-hole he first gets the light upon the eye; this is best effected from a short distance away. When the light is well on the pupil and the observer can see the red reflex he approaches slowly nearer and nearer, watching that the light does not leave the pupil, until his brow is almost or quite touching the patient's brow. If now both the patient and theobserver are emmetropic, the inexperienced observer will probably see the details of the fundus only indistinctly. He should then turn up with his index finger applied to the milled disc on the back of the ophthalmoscope successively stronger concave glasses (usually marked with white numerals). He will then probably see the fundus quite clearly. The image is erect, *i.e.*, the opposite of that by the indirect method. Here again practice is needed, and a knowledge of the optical conditions is quite essential.

We will now consider the chief features which are to be learnt in each stage of the examination, and how they are to be learnt.

I. Preliminary Examination with the Mirror at 1 metre. We will suppose that the observer is emmetropic, or that his refraction has been corrected, and that the accommodation of the observed eye is at rest or paralysed. In examining the right eye the patient is told to look at the observer's right little finger which is held up; this is easily done while holding the ophthalmoscope. In examining the left eye the patient is told to look at the observer's left ear. In this manner the optic disc, which lies a little to the nasal side of the posterior pole of the eye, is brought into the observer's line of vision. When the optic disc is opposite the pupil we shall notice from a distance of 1 metre that the red reflex becomes paler or even whitish.

If the eye is highly hypermetropic or myopic we shall see some details of the fundus, *e.g.*, a few vessels running across the reflex. The explanation is easy from what we have already learnt.

Consider first the hypermetropic eye. If we think of two spots on the retina, say at opposite edges of the disc, the rays reflected from these points will form two bundles of divergent

rays when they leave the eye, just as if they came from the corresponding virtual remote points behind the eye (Fig. 79). The greater the distance from the eye, the greater will be the area over which these divergent rays will spread, so that at 1 metre some of the peripheral rays of each pencil will enter the observer's eye. By a slight effort of accommodation the observer will be able to bring these divergent rays to a focus on his retina, so that he will obtain a clear image of each point, and necessarily also of the intervening region. It is exactly as if the eye were taken away and the two points were situated at the remote points. Hence the image will be erect.

If the observer now shifts a little to one side, the observed eye remaining stationary, more rays will enter his eye from the neighbourhood of the opposite point, and less from the

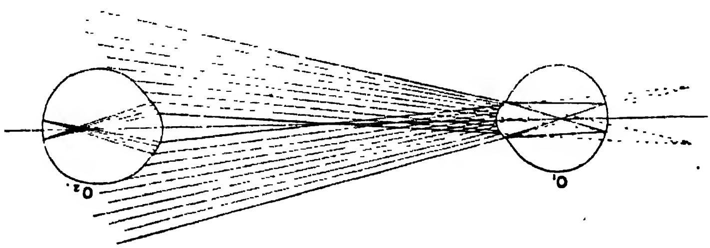


FIG. 79.—Examination with the mirror at 1 metre. O_1, observed eye, which is hypermetropic ; O_2, observer's eye, emmetropic, but accommodated for the divergent rays from O_1.

neighbourhood of the point on the same side as that to which his movement is directed. Although the points remain stationary, more of the fundus on the opposite side and less of the fundus on the same side will be seen. Hence the points will seem to move in the same direction as his own movement. The observer mentally regards the very sharp outline of the pupil as a fixed object of comparison, and as more of the fundus on the opposite side comes into view, whilst a corresponding amount on the same side disappears, this is mentally interpreted as a movement of the image in the same direction.

If, therefore, when the light is reflected into the eye at a metre distance we see vessels in the pupillary reflex, and if they appear to move in the same direction when the head is moved slightly to one side, we conclude that the eye is hypermetropic.

Consider now the myopic eye (Fig. 80). Here the emitted rays from the two points will be strongly convergent in each case, and a real inverted image of the points and intervening

area will be formed at the remote point of the eye, *i.e.*, between the observer and the observed eye. The rays will diverge from this image, and the effect will be exactly the same as

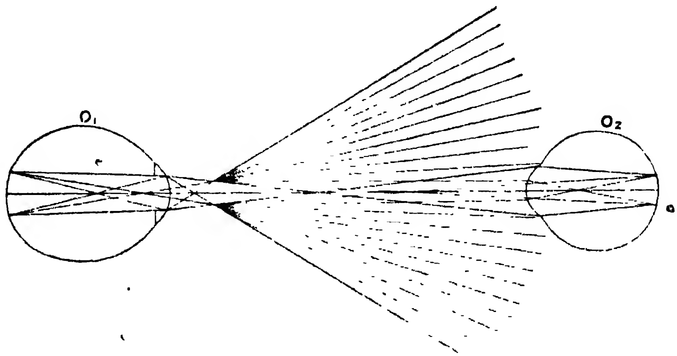


FIG. 80.—Examination with the mirror at 1 metre. O_1, observed eye, which is highly myopic; O_2, observer's eye, emmetropic, but accommodated for the divergent rays from the far point of O_1.

if there were an actual inverted object in this position. If the myopia is sufficiently high, the image will be beyond the observer's near point, so that he will be able to accommodate for it. If he moves to one side he will see more of the observed

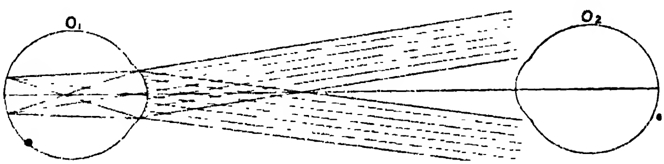


FIG. 81.—Examination with the mirror at 1 metre. O_1, observed eye, which is emmetropic; O_2, observer's eye: none of the rays from the widely distant points on the fundus of O_1 enter O_2. If the points are close together the rays of the two bundles will be nearly parallel, and would form a clear image on the retina of O_2 if the accommodation of O_2 were almost completely in abeyance.

fundus on the same side and correspondingly less on the opposite side, so that the fundus will appear to have moved in the opposite direction.

If, therefore, when the light is reflected into the eye at a metre distance we see vessels in the pupillary reflex, and if they appear to move in the opposite direction when the head

is moved slightly to one side, we conclude that the eye is myopic.

What will happen in emmetropia (Fig. 81) or low myopia, for the effect will be similar? Here the rays passing out of the eye from the two points will be parallel or very slightly convergent, and their direction will be that of their axes, which is the continuation of the lines joining the points with the nodal point of the eye. As these axes constantly diverge from one another, the observer at a distance of 1 metre cannot receive portions of both pencils of rays upon his own pupil, consequently he cannot obtain a clear image of the whole intermediate region between the spots. He may get a clear image from two spots very close together, but only if his accommodation is almost completely suspended, so that nearly parallel rays are brought to a focus upon his retina.

The same reasoning applies to low hypermetropia, for here the remote point of the eye is so far behind the retina that the rays diverge very little when they leave the eye. so that they are almost parallel.

If, therefore, when the light is reflected into the eye at a distance of a metre we see only a red reflex in the pupil, without any details, we conclude that the eye is either emmetropic or has only a low degree of ametropia.

A still simpler means of discovering the condition of the refraction is as follows. Still throwing the light into the eye with the large concave mirror we tilt it gently in various directions. We shall see a shadow move across the pupil; if the shadow is very dark there is considerable error of refraction. If it moves in the opposite direction to that in which we move the (concave) mirror the eye is hypermetropic; if in the same direction it is myopic. This method is used for correcting refraction, and we shall consider it in detail later (*see* Retinoscopy).

II. Preliminary Examination with the Mirror at the convenient distance for near vision (22 cm.). At this distance the observer will be most suitably situated for distinct unaided vision, and he will be able to examine the superficial parts of the eye more accurately. If he is presbyopic he will naturally have to correct his presbyopia, and he may have to use a convex lens if he is strongly hypermetropic. If he is very myopic he will have to approach closer.

The advantages of a preliminary examination in this manner are (1) the recognition of opacities in the refractive media; (2) the recognition of a detached retina or other substance not

far behind the lens ; (3) the confirmation of the results found by the external examination.

(1) *The diagnosis of opacities in the refractive media.* If the eye is normal there will be a red reflex from the pupil. If there is any opaque body in the course of the rays reflected from the fundus it will stop these rays and will therefore appear black. The whole field may be black, as when the lens is entirely opaque, or when there is blood in the vitreous. In the latter case oblique illumination will show the red blood behind the transparent lens if it is sufficiently far forward in the vitreous : the blood looks red in this case because of the light reflected from its surface.

Opacities vary in shape, size, and position. We are particularly concerned to discover their position, as this frequently gives the key to their nature.

The first point to determine is whether the opacity is movable. This is done by telling the patient to move his eye in different directions—towards the ceiling, towards the floor, to the right, to the left—and then to look straight forward. A floating opacity will then continue to move after the eye is brought to rest. It must therefore be either in the aqueous or vitreous. In the former position it can be seen and diagnosed by other methods. If it is in the vitreous and is freely movable we also learn that the vitreous is fluid, which is not its normal consistency. If the opacity moves only with the eye it may be in the cornea, lens, or vitreous, which, under these circumstances, will have its usual viscous consistency.

The next point is to determine its exact position. This is effected in the preliminary examination with the mirror alone by *parallactic displacement.*

In Fig. 82, if 4 is the centre of rotation of the eye, and if there are opacities at 1, 2, 3, 4, 5, then, when the eye is rotated a small amount, the opacities, 1, 2 and 3, in front of the centre of rotation will move in the direction of rotation, and 5, behind the centre, will move in the opposite direction, while 4, at the centre, will not move. It is obvious that the amount of movement will be greater the farther the opacity is from the centre of rotation. Now, we have no means of defining the centre of rotation by ophthalmoscopic examination, but all the movements will be referred to the edge of the pupil for comparison (*vide* p. 105). If the observer is situated at A, all the opacities will appear as a single spot in the centre of the pupillary reflex. If he shifts his position to B, or if the eye is rotated a corresponding amount in the opposite direc-

tion, the opacity 2 will remain in the centre of the pupil, whilst 1 will appear to move towards one edge of the pupil, and 3, 4 and 5 towards the opposite edge, 5 being lost entirely behind the iris.

Hence we deduce the rule that if the eye is moved slightly in a given direction, opacities in the pupillary plane will appear stationary; those in front of that plane will move in the same direction, and those behind will appear to move in the opposite direction, the amplitude of apparent movement being a rough indication of their distance from the pupillary plane.

There is another guide which we may make use of, viz., the corneal reflex. This is the image of the mirror formed by

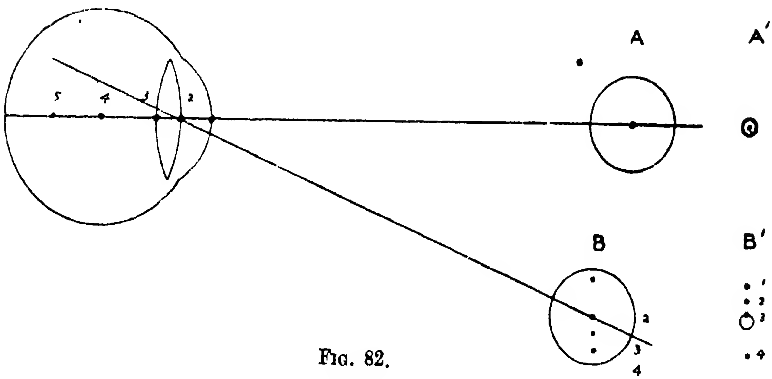


Fig. 82.

the cornea. With the ordinary convex mirror it is a virtual image (*vide* p. 26) situated about 4 mm. behind the anterior surface, *i.e.*, a short distance behind the anterior surface of the lens (behind 2 in Fig. 82). The centre of curvature of the cornea is situated 8 mm. behind its anterior surface, *i.e.*, less than 1 mm. behind 3 (Fig. 82). The corneal reflex will always cover this latter spot, the centre of curvature of the cornea, no matter what the position of the eye. Hence an opacity situated here will always be covered by the corneal reflex; opacities in front of the centre of curvature move in the same sense with regard to the reflex as the eye moves; and opacities behind it move in the opposite direction to the movement of the eye. Therefore, in Fig. 82, in the first position of the eye, the opacities 1, 2, 3, 4, 5, will all appear in the centre of the corneal reflex (A'); in the second position they will appear as in B'; so that an opacity at the posterior

pole of the lens will scarcely leave the edge of the reflex, whereas an anterior polar opacity will move much farther from it.

One peculiar apparent opacity is seen by the mirror alone, and this method affords the surest means of discovering the defect. This is the edge of a dislocated lens, or the notch in the edge of the lens in congenital coloboma of the lens. When the edge of the lens crosses the pupillary area it is seen as an intensely black crescent, sharply defined peripherally but merging centrally into the clear red reflex. The reason of this appearance is that the whole of the light reflected from the fundus which falls upon the extreme edge of the lens is totally reflected within the lens; none of it leaves the eye, so that none can enter the observer's eye.

We not infrequently meet with very fine opacities, especially in the vitreous. If we use a concave mirror and a bright light we shall probably fail to see them, the reason being that these very delicate opacities are partially transparent, so that if the light is very bright some passes through them and contrast is reduced. Contrast is further reduced by reflection of light from their surfaces. They are, as it were, drowned in light. In order that we may be sure of not missing fine opacities the best method to adopt is to use a plane mirror. The rays reflected from a plane mirror are divergent (*vide* p. 25); hence less light enters the eye. If we have no plane mirror available the light should be reduced, but this is not so satisfactory. We may increase our chances of seeing the specks if we place a convex lens behind the mirror, which will have the effect of magnifying them (Fig. 21).

(2) *The recognition of detached retina, &c.* A detached retina is situated much in front of its normal position; it is therefore in the position of the fundus of a very hypermetropic eye. When light is thrown in from a mirror at reading distance light will probably be reflected from the surface in such a way that some rays will enter the observer's eye. A difference of reflex in different directions is noticed, red in some, grey or black in others. More minute investigation will reveal a whitish or greyish uneven surface upon which there are almost black wavy lines; these are the retinal vessels. Particular stress is laid upon this point, because the appearances of a detached retina by the indirect and direct methods may be very puzzling to the beginner. If the precaution is always taken of commencing the examination with the mirror alone little difficulty is likely to arise.

Besides a detached retina we shall also be able to see anything else in a similar position, *e.g.*, a tumour pushing the retina forwards, or a tumour of the retina itself (glioma), and so on.

None of these will be seen unless they are pushed forwards very considerably ; hence we must not asseverate the absence of a detached retina, &c., if we fail to see it by this method.

(3) *Confirmation of the results found by the external examination.* We are able by this method not only to confirm the results previously arrived at by external examination, but also to supplement them by important subsidiary information. Thus we are able to map out the limits of opacities in the lens much more accurately, since they now appear black on a red background, and as has already been shown we can determine their exact position with much greater precision.

We may have noticed a black spot in the iris in a case with the history of a foreign body having gone into the eye. It is probable that the foreign body has passed through the iris, and that the black spot is a hole. The examination with the mirror often at once settles the question, for if there is a hole we shall be able to find some position in which a red reflex can be seen through the hole. The absence of a red reflex does not prove the absence of a hole, for the lens may be opaque behind the hole.

The following is a somewhat similar example. We have noticed a black patch at the ciliary margin of the iris, convex in outline towards the pupillary margin. It may be a melanotic sarcoma of the ciliary body growing forwards and implicating the iris : or it may be a separation of the iris from its ciliary attachment (iridodialysis). In the latter case it will be possible to obtain a reflex through it by the mirror, whereas in the former it will be opaque.

We have said that by this method opacities in the refractive media appear black. Superficial opacities, however, such as those in the cornea and near the anterior surface of the lens, can be seen in their natural colours by approaching still nearer to the eye. Under these conditions more light is reflected from the surface of the opacities and some of it enters the observer's eye. It will be objected rightly that now we shall be within our near distance and consequently shall not be able to see anything clearly. This is true, but it can be obviated by assisting our accommodation by putting up gradually stronger convex glasses behind the ophthalmoscope mirror as we approach the eye. This has the additional

advantage of magnifying the opacity. If we approach very close to the eye and place a + 20 D lens behind the mirror we shall see the opacities highly magnified. This glass will be acting very much like an ordinary magnifying glass, so we shall have to focus it in much the same manner. We therefore start a little distance from the cornea and watch carefully as we get nearer and nearer; there will come a point when the opacity is very clearly defined.

Suppose now that under the same conditions, with the + 20 D lens in the position for seeing the cornea, we wish to examine an opacity near the surface of the lens it can be done in two ways. We may continue to approach still nearer until it comes into focus; or we can use a weaker lens, retaining our

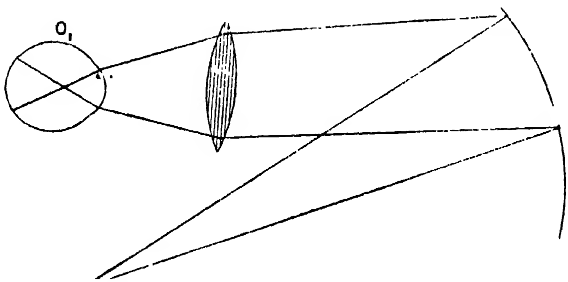


FIG. 83.—Indirect method. Illumination of the fundus, showing the course of rays from the source of light to the mirror, through the lens, and through the eye; also the area of the field of illumination.

original position. The weaker lens is most easily produced by moving up stronger and stronger concave lenses in front of the + 20, until the opacity is accurately focussed. This is made possible in most ophthalmoscopes by having a + 20 D lens set in a disc which lies behind the sight-hole: the lens can be turned into position when required, and does not interfere with the use of the other lenses at the same time. The opacity in the crystalline lens will, of course, not be quite so highly magnified by the second as by the first method.

III. The Indirect Method. The indirect method of examination with the ophthalmoscope consists essentially in making the eye, whatever be its refraction, highly myopic by placing a strong convex lens in front of it (Figs. 83–85). The effect of this will be to form a real inverted image of the fundus between the observer and the convex lens, as will be

easily understood from the accompanying diagrams. If the eye is already myopic the convergent rays which come from any point on its fundus will be made still more convergent by the lens, and the inverted image which is always formed in myopia will be brought close to the lens. If the eye is emmetropic the parallel rays emitted will be made strongly convergent, and where they cross the inverted image will be formed. If the eye is hypermetropic the rays will still be made convergent, for the lens used is so strong that the divergence in hypermetropia is never strong enough to prevent it.

It will be seen that with the same lens the inverted image is formed at different distances beyond it according to the refraction of the eye. If the lens is kept at a constant distance from the eye, *e.g.*, its own focal distance, the emmetropic image will be formed at the focal distance of the lens beyond it: the myopic will be nearer to the lens, the hypermetropic farther from it (Fig. 85).

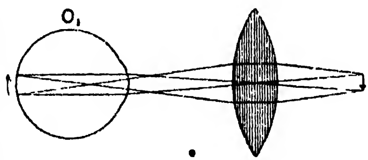


FIG. 84.—Indirect method. Emergent rays from the fundus, showing the formation of the image. In the figure the lens is situated at the anterior focal plane of the eye; the rays which are parallel inside the eye, therefore, pass through the optical centre of the lens. The rays which pass through the nodal point of the eye are rendered convergent by the lens. The points where these two systems of rays cross give the position of the image, which is seen to be inverted.

In all cases the image is magnified, the amount of magnification depending upon the refraction of the eye, the strength of the lens, and its distance from the eye. With a + 13 D the fundus of an emmetropic eye is magnified about five times.

One of the greatest difficulties in using the indirect method is the group of reflexes formed by the eye and the surfaces of the lens. We have seen that the cornea forms a reflex of the mirror when it is used alone. This reflex, when seen through the convex lens, is magnified, so that it may cover the pupil and prevent anything behind being seen. But the surface of the lens towards the observer acts like another convex mirror and forms another reflex situated behind the lens. Similarly the surface of the lens near the patient acts like a concave mirror and forms a reflex on the observer's side of the lens. These reflexes are very troublesome, but they may be got out of the way by a little manœuvring. It has been said that the two lens reflexes, which are the most troublesome, are images

of the mirror formed on opposite sides of the lens. If we tilt the lens a little it will be found that these reflexes move in opposite directions, and we can look quite comfortably between them. We must be careful not to tilt the lens more than is necessary, because if we look obliquely through a tilted lens objects appear distorted: in fact we produce one type of astigmatism. The distorted image of the disc produced in this manner may be attributed to astigmatism in the eye, when none is really present.

Another difficulty which the beginner usually experiences is due to getting too close to the patient. If he understands the position of the image which he is looking for, as described above, he will discover why he can see no sharp image when

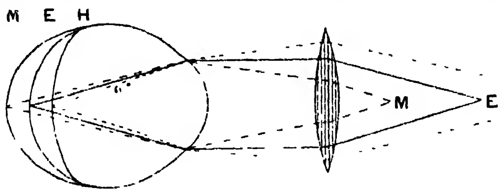


FIG. 85.—Indirect method. Position of the image according to the refraction of the eye. In this figure the lens is situated at its own focal distance from the cornea. In emmetropia the parallel emergent rays, therefore, cross at the principal focus of the lens E. In myopia the convergent emergent rays cross nearer to the lens than its principal focus, viz., at M; in hypermetropia the divergent emergent rays cross farther from the lens than its principal focus, viz., at H.

he is too close to the patient. Most people using the indirect method think that they are looking at the pupil. As a matter of fact, when the fundus is seen clearly, they are not accommodating for the pupil, but for the real image of the fundus, which is in the air somewhere between the lens and the observer. Now we can only see an object clearly with the unaided eye if it is at a convenient distance away. Consequently, if the observer gets so close to the patient that he is less than the distance of his near point from the aerial image he cannot see it clearly.

If we like to do so we can get over this difficulty and still remain closer to the patient. In order to do so, the observer must help his accommodation by putting up a convex lens in front of his eye. If we put up a + 1 D or + 2 D behind the ophthalmoscope mirror, we shall not only see the image clearly at a shorter range, but we shall also magnify it, an additional advantage.

As regards the position of the convex lens before the patient's eye, there is a considerable range over which we can see the fundus quite well, but some positions are better than others. In practice we find the best position by putting the lens close to the eye to start with, steadying it by the little finger applied to the patient's brow, and gradually bringing it farther away from the eye until the best position is obtained.

Theoretically, from the point of view of the maximum field of fundus seen, the best place for the lens is its own focal distance from the patient's pupil. But this is the very worst place from the point of view of the corneal reflex. The latter is situated near the level of the iris (4 mm. behind the cornea, *vide* p. 109). If the convex lens is at its focal distance from it, the rays from this image will be made parallel by the lens, *i.e.*, the reflex will fill the whole area of the lens, and we shall see nothing else. Hence the best position for practical purposes is either nearer to or farther from the eye than this position. We shall see later that a convenient distance is when the lens is at its focal distance from the anterior focus of the eye. Here, slight tilting of the lens, besides shifting the lens reflexes out of the way, will also move the corneal reflex and the image of the fundus in opposite directions, and so get the corneal reflex out of the way.

We can tell by the indirect method whether the eye is emmetropic or ametropic by observing the effect of shifting the lens on the size of the image of the fundus. We use the disc as the best guide, getting it into the field by telling the patient to look in the appropriate direction, *i.e.*, towards the raised right little finger when examining the right eye, towards the left ear when examining the left.

Place the lens close to the eye and gradually bring it farther away. If the image of the disc does not alter in size the eye is emmetropic; if it gets smaller the eye is hypermetropic; if it gets larger the eye is myopic.

If we understand why this is so, we shall be able to remember what happens in each case. Imagine two points, *a* and *b*, upon the fundus, *e.g.*, upon opposite edges of the disc (Fig. 86). If they are illuminated, one of the many divergent rays emitted by each must be parallel to the axis. These two rays, when they pass out of the eye, will cross at the anterior focus of the eye, whatever its refraction may be, as long as the error is one of undue length or shortness (*axial ametropia*) They will cross at the anterior focus because they are parallel to each other before refraction. Now, suppose that the convex lens used in the

indirect method is situated at its focal distance from the anterior focus of the eye. These two rays, after they have crossed, will meet the convex lens, and since they come from the focus of the lens they will be parallel to each other after refraction. As we have already seen (p. 113), under such circumstances the image of the fundus is formed in emmetropia at the focal distance of the convex lens (F^1) from it (E): in myopia the image will be nearer the lens (M), in hypermetropia farther away (H). The two rays which we have been considering must represent the images of the two points on the fundus. Hence the distance between them after refraction by the eye and by the lens will give the size of the image of the portion of fundus between *a* and *b*. We see therefore that when the lens is at its own focal distance from the anterior focus of the eye the magnification of the image

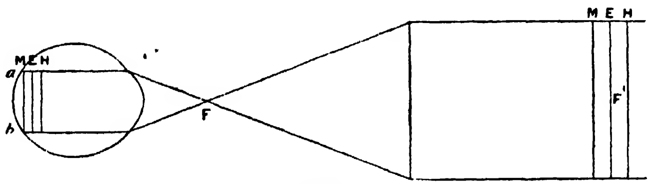


FIG. 86.

is the same in emmetropia, in axial myopia, and in axial hypermetropia.

If the lens is nearer the eye than the above distance (Fig. 87), the rays under consideration will diverge after refraction by the convex lens. Hence, if the lens is less than its own focal distance from the anterior focus of the eye, the magnification is greatest in axial hypermetropia, least in axial myopia, and intermediate in emmetropia. Conversely, if the lens is farther from the eye, the rays under consideration will be convergent after refraction by it. Hence, if the lens is more than its own focal distance from the anterior focus of the eye, the magnification will be greatest in axial myopia, least in axial hypermetropia, and intermediate in emmetropia.

In *curvature ametropia*, such as we meet with in astigmatism, the results are not quite the same, but they are easily deduced if we remember that there are now two anterior foci to the eye, one for each meridian. There are also two nodal points. When the lens is at its focal distance from the cornea the magnification is the same in emmetropia and any ametropia of curvature. In these circumstances the disc appears circular. If the lens is nearer the eye the image is elliptical, with its long axis in the less refractive meridian, *i.e.*, generally horizontal. If the lens

is farther from the eye the long axis is in the more refractive meridian, *i.e.*, generally vertical. As mentioned before, it is essential that the lens should be held almost vertical, as any inclination makes it itself astigmatic. If the disc is really oval, as in high myopia, the axis of the ellipse will of course remain unaltered.

Ametropia of index of refraction occurs in old age. Aphakia, the condition when the lens has been removed, may be considered an extreme form of index ametropia. Here the position of the nodal point remains invariable, and if the convex lens is at its focal distance from this point the image is the same size in emmetropia and ametropia of index of refraction. If the lens is moved closer to the eye the image increases in hypermetropia and diminishes in myopia, while it remains the same in emmetropia.

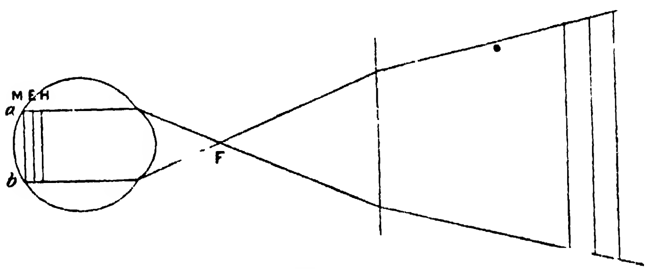


FIG. 87

Since the image is formed at a considerable distance beyond the focus of the lens in the high hypermetropia of aphakia it is convenient to use a stronger lens, *e.g.*, + 18 D.

Differences of level of two points near each other on the fundus are made very evident by parallactic displacement in the indirect method. Thus, in Fig. 88, if there are two spots, *a* and *b*, at different levels in the fundus, *e.g.*, on the edge of the disc and at the bottom of a glaucoma cup, when the lens is shifted slightly so that its optical centre moves from o_1 to o_2, the images of *a* and *b* will move from a_1 to a_2 and b_1 to b_2. It is of historical interest that this displacement was at one time wrongly interpreted, so that a glaucomatous cupping of the disc was diagnosed as a swelling.

IV. The Direct Method. In the direct method the observer approaches as close as possible to the patient's eye (Fig. 89). If the eye is hypermetropic the emergent rays will be divergent, as if coming from the virtual remote point behind the

eye. Owing to the short distance between the eyes a large pencil will fall upon the observer's pupil, and may be brought to a focus upon his retina if he makes a suitable effort of accommodation. If he is presbyopic, or if his accommodation is relaxed, he will only obtain a clear image by placing a convex lens behind the sight-hole of the mirror (Fig. 90, H).

If the observed eye is emmetropic the emergent rays will be parallel, and consequently can only form a clear image upon the observer's retina if his accommodation is absolutely relaxed (Fig. 90, E)—unless, indeed, he counteracts the amount of his accommodation by a corresponding concave lens in front of his eye (*vide* p. 119).

If the observed eye is myopic the emitted rays are convergent. If the myopia is moderate the real image of the fundus at the far point of the eye will be behind the observer's head, *i.e.*, he will catch the convergent rays before they have come to

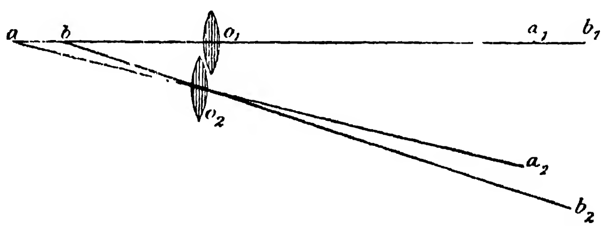


FIG. 88.—Indirect method. Parallactic displacement.

a focus. These convergent rays, entering his emmetropic eye, are brought to a focus in his vitreous ; hence he cannot possibly obtain a clear image unless he counteracts the convergence by an equivalent concave lens behind the mirror (Fig. 90, M). If the observed eye is very highly myopic its punctum remotum will be situated somewhere in the space between the eye itself and the observer's retina, and it may be in such a position that it is impossible to obtain a clear image with any correction. For example, the remote point may be just behind the sight-hole of the mirror. Here it is too close to be accommodated for, and no correcting glass situated at the same position will have any effect upon the rays, for they will nearly all pass through the optical centre of the lens. The practical outcome of this discussion is to get as close to the eye as possible.

Much stress is generally laid upon the necessity and the difficulty of relaxing one's accommodation in examination by the direct method. It is difficult to relax the accommodation

entirely when the eye is apparently close to the object looked at. The observer should try to think that he is looking at a very distant object, but even then, as soon as he directs his attention to details of the picture, he is almost certain to accommodate. It is best for the beginner not to worry himself about this point: if he cannot see an emmetropic fundus clearly let him put up minus lenses until he does. After he has acquired facility in seeing anything at all it will be soon enough for him to grapple with this difficulty.

The image by the direct method is always erect. Thus, in hypermetropia it is exactly as if the observer were looking at an actual object situated at the remote point of the eye, *i.e.*, some distance behind the eye. In myopia the converging

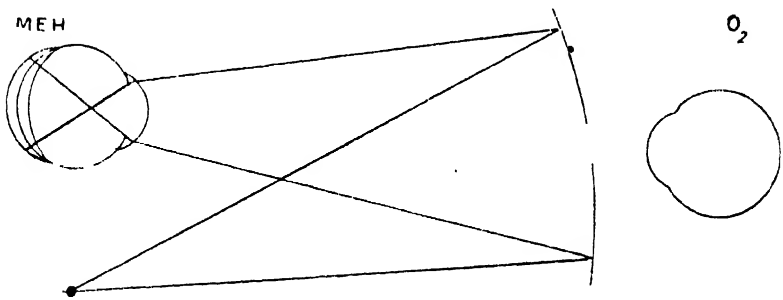


FIG. 89.—Direct method. Illumination of the fundus, showing the course of rays from the source of light to the mirror and through the eye; also the area of the field of illumination. Compare with Fig. 83.

rays are caught before they cross; they are made suitably less convergent by the correcting lens behind the mirror, so that again an erect image is seen. In emmetropia the emergent rays are parallel and are, therefore, also caught before they cross—at infinity; hence, again, an erect image is seen.

The image is always magnified, and it is magnified more than by the indirect method. In emmetropia the fundus is seen magnified about fifteen times. In hypermetropia it is magnified less, and in myopia more than in emmetropia.

The field of ophthalmoscopic vision by the direct method, *i.e.*, the area of the fundus which can be seen, varies with the distance of the observer from the eye and with the refraction of the eye. It increases as the eye is approached—another reason for getting as close to the eye as possible. It is greatest in hypermetropia, least in myopia, and intermediate in emme-

tropia. Thus, we see the largest area, least magnified, in hypermetropia, and we see the least area, most magnified, in myopia.

In astigmatism the magnification is greatest in the more myopic meridian, and least in the more hypermetropic. In the usual form of astigmatism the image of the disc is an ellipse with the long axis vertical—the opposite of the usual image by the indirect method, with the lens near the eye (*vide*

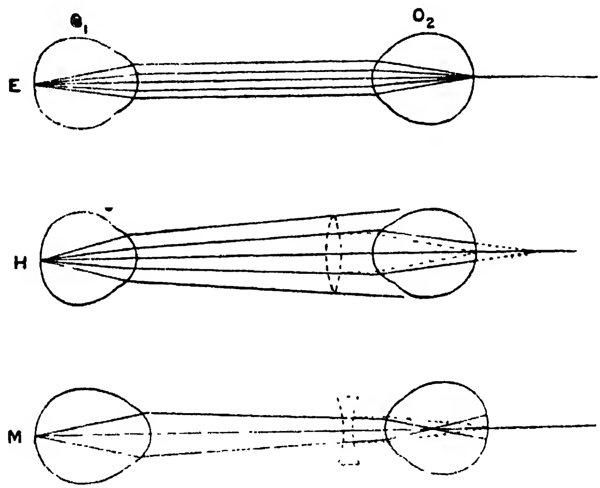


FIG. 90.—Direct method. Emergent rays from the fundus of the observed eye, O_1, showing the formation of the retinal image on the retina of the observer's eye, O_2. In emmetropia, E, the emergent parallel rays are brought to a focus on the retina of O_2 if the accommodation of this eye is absolutely at rest. In hypermetropia, H, the emergent divergent rays are brought to a focus on the retina of O_2, either by means of accommodation or by placing a convex lens in front of O_2. In myopia, M, the emergent convergent rays can only be brought to a focus on the retina of O_2 by placing a concave lens in front of O_2.

p. 116). It is obvious that there can be no clear image of the whole field by the direct method in astigmatism. *Only lines perpendicular to the meridian which is corrected are seen clearly.* Lines in any meridian other than the two principal ones cannot be seen clearly by any spherical correcting glass, but only by a cylindrical lens or combination of a spherical and a cylindrical.

From what has been said it is obvious that the correcting lens behind the sight-hole of the mirror also represents the

spectacle glass which will be required to correct the refraction if it is placed in the same position. This, of course, is only true if the observer is emmetropic, or has his refraction corrected, and if his accommodation is quite at rest. Since it is difficult to relax the accommodation completely, this method of determining any error of refraction should only be used as a rough estimate, unless the observer is an expert.

If there is a difference in level between two points on the fundus, *e.g.*, the edge of the disc and the bottom of a glaucoma cup, it is made manifest by the direct method also by parallactic displacement. If we focus the edge of the disc and then move slightly to one side the edge of the disc will appear to move over the bottom of the cup, *i.e.*, it moves in the opposite direction. An object farther forward, therefore, moves in the opposite direction to the movement of the observer's head.

The difference in level can be accurately measured. In the example given, the bottom of the disc will be relatively myopic to the edge, since it is farther away from the back of the lens. If the eye is emmetropic and the edge of the disc can be seen clearly without the assistance of any correcting lens we shall require a concave lens to see the vessels at the bottom of the cup clearly. It can be proved that *if the correcting lens is at the anterior focus of the eye* a difference of 3 D is equivalent to 1 mm. difference of level. We must get as close as possible to the eye when measuring differences of level, because only then are the conditions of accuracy fulfilled. If, with the observer's accommodation at rest, he sees the edge of the disc clearly with no correcting lens, but requires — 3 D to see the bottom clearly, he knows that the bottom is 1 mm. behind the edge. He will of course see the bottom clearly with a higher concave lens if he counteracts the excess by accommodating: hence he must be careful to choose the lowest minus lens.

Similarly projections forwards can be measured. Here the observer chooses the highest convex lens with which he can see some well-defined point on the top of the eminence. The same rule that 3 D is equivalent to 1 mm. holds good. Suppose, for example, that he is measuring the swelling of the disc in a case of papillœdema. He first finds the highest convex glass with which he can see clearly a retinal vessel a little distance away from the disc. He then finds the highest convex glass with which he can see a vessel or a small hæmorrhage as near the top of the swollen disc as he can judge. The

difference between the two lenses will give the height of the swelling.

It will be seen that the difficulty of relaxing the accommodation enters into this estimation. The student need not, however, be dismayed. Very fairly accurate results can be obtained without relaxing the accommodation, for if he is accommodating the same amount when he measures the top of the swelling that he is when he measures the level of the surrounding fundus the difference between the two observations will be the same as if he was not accommodating at all. He can ensure this fairly well by always choosing the highest convex lens; it is safest to choose the lens which just makes the object looked at appear a little blurred. Of course, the eye may be myopic; if for "convex lens" in the above description "relatively convex lens" be substituted (— 1 D being relatively convex to — 4 D) the principle is the same.

An object in the vitreous, *e.g.*, a large opacity, is in the same condition as the fundus of a hypermetropic eye. If the eye is emmetropic, so that the fundus is visible without any correcting lens, the opacity can be examined either by accommodating for it or by putting up convex lenses until it is clearly focussed. If it is close behind the lens, accommodation will have to be assisted by a convex lens in any case, unless the observer withdraws farther from the eye. It will be seen, therefore, that by putting up convex lenses from 0 to + 20 D we can thoroughly explore the emmetropic eye from the fundus to the surface of the cornea.

Examined in this manner the appearance of opacities in the vitreous or lens will vary with the amount of light stopped by them, *i.e.*, by their density, and with the amount of light reflected from their surfaces. If they are very dense they will appear black against the background of the red reflex. If they are semi-transparent they will appear red or whitish according to the relative amounts of light transmitted from the fundus and reflected from the surface (*v.* p. 110). A detached retina may therefore look red or white, according to its degree of transparence. If much light is reflected from the surface details may be seen upon it; otherwise it appears uniformly black.

CHAPTER VIII

The Fundus Oculi

When the fundus is observed by the indirect method it is seen to be of a bright red colour. This is due chiefly to the blood circulating in the choroid. In people of dark complexion no choroidal blood vessels are seen on account of the retinal pigment epithelium, which, while dense enough to blur any details, is not sufficiently so to prevent the colour of the blood manifesting itself.

The Optic Disc. The first object to be sought is the optic disc or papilla (Plate III., Fig. 1). As already mentioned, it is done by making the patient look slightly towards the nasal side. The reflex then suddenly changes from bright red to pale red, and if the optical conditions are properly arranged in accordance with the directions given in the last chapter the disc will be clearly seen.

The disc is pale pink in colour, the tint showing considerable variations within the limits of normality. It is nearly circular but seldom perfectly so; it is about 1·5 mm. in diameter, but of course is seen magnified. The oval appearance due to astigmatism must be borne in mind (*vide* p. 120). The edges are usually quite sharp, but sometimes a little irregular. Not uncommonly, especially in old people, there is a narrow white ring around the pink disc, the *scleral ring;* this is due to the choroid and the pigment epithelium of the retina not extending quite up to the margin of the disc so that the sclerotic is seen through the retina. Sometimes there is a ring of black pigment around the margin of the disc, due to the retinal pigment epithelium being heaped up here. More commonly parts of the circumference have black patches, but they are not continuous. These features are of no importance from the pathological point of view.

The disc itself is seldom uniformly pink. The central part is usually paler and may be quite white, and this lighter area may extend nearly to the temporal edge of the disc; it rarely extends quite to the edge. The temporal side is therefore normally paler than the nasal. The central vessels emerge

from the middle of this white area, and careful examination with the direct method will show that the area is a funnel-shaped depression, the *physiological cup*. This cup varies very much in different eyes. When it is very deep the central part may be seen to be speckled with grey spots; these are the meshes of the lamina cribrosa through which the nerve fibres are passing. Sometimes there is scarcely any physiological cup; the disc is then more uniformly pink, and the central vessels may have already divided before they come to the surface. The true nature of the physiological cup is best understood by comparing the ophthalmoscopic picture with a microscopic section vertically through the nerve head.

The colour of the disc is due to the white fibres of the lamina cribrosa seen through the vascularised nerve fibres. Where the nerve fibres are thinnest, *i.e.*, in the cup, the white lamina shines through brightest. The grey spots in the lamina, when they are seen, are due to the non-medullated nerve fibres reflecting less light than the white connective tissue fibres.

The Retinal Vessels. The retinal vessels are derived from the central artery and vein, which usually divide into two branches at or near the surface of the disc. These branches are above and below, and form a superior and an inferior trunk (Plate I.). Each trunk usually divides into two, one of which sweeps up or down towards the temporal side, the other sweeping up or down towards the nasal side. These branches are called the superior and inferior temporal and nasal arteries and veins. They divide dichotomously into innumerable branches.

The arteries are distinguished from the veins in being lighter red and narrower. The veins have a purplish tint and are often more convoluted; less frequently the arteries are tortuous. What is seen is the blood column, not the actual vessel wall, which is transparent. Each, but especially the arteries, may have a bright silvery streak running longitudinally down the centre, due to reflection of light from the convex cylindrical surface.

The mode of branching of the vessels is subject to great variation, though it is derived from the fundamental type described. The variations are generally of no practical importance. The primary division of the superior and inferior trunks usually takes place on or very near the disc. The nasal branches run much more radially than the temporal, which make a very decided sweep to avoid the macula.

PLATE III.

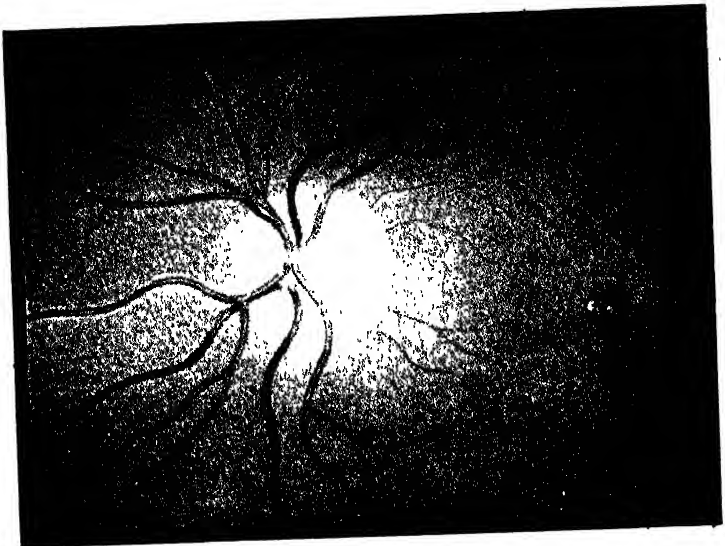


Fig. 1.—Normal fundus.

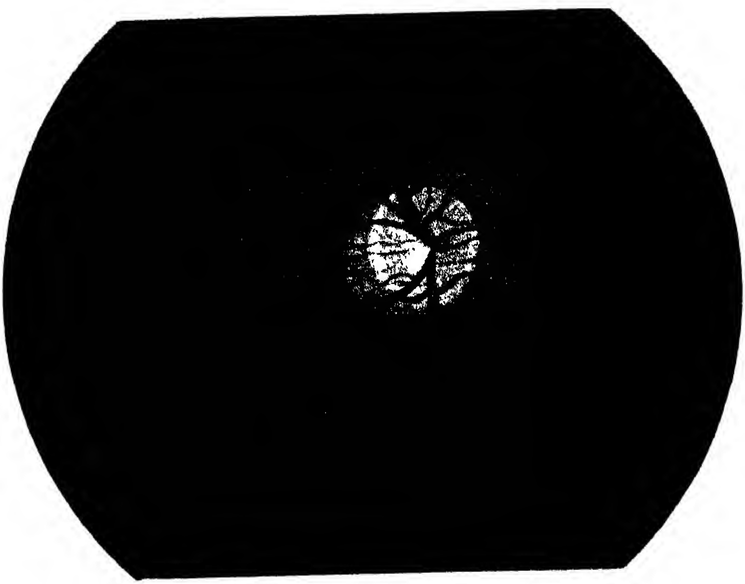


Fig. 2.—Normal fundus: "tigroid" variety.

[*To face p* 124.

PLATE IV.

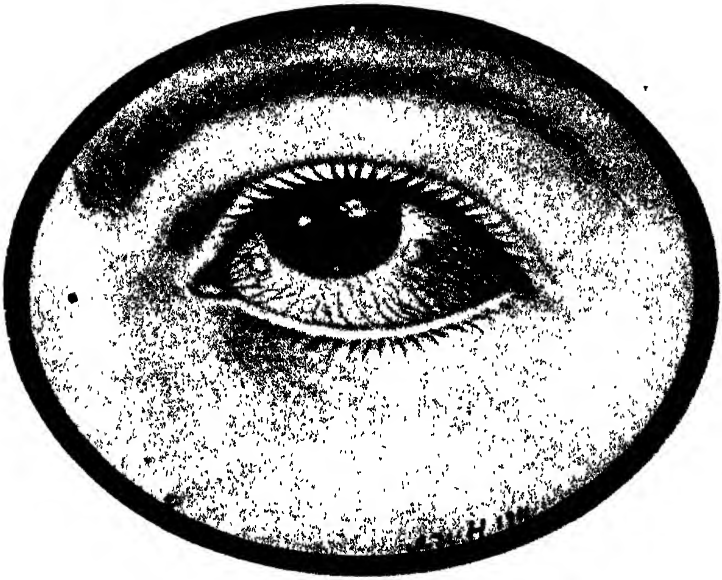


FIG. 1. Phlyctenular conjunctivitis.

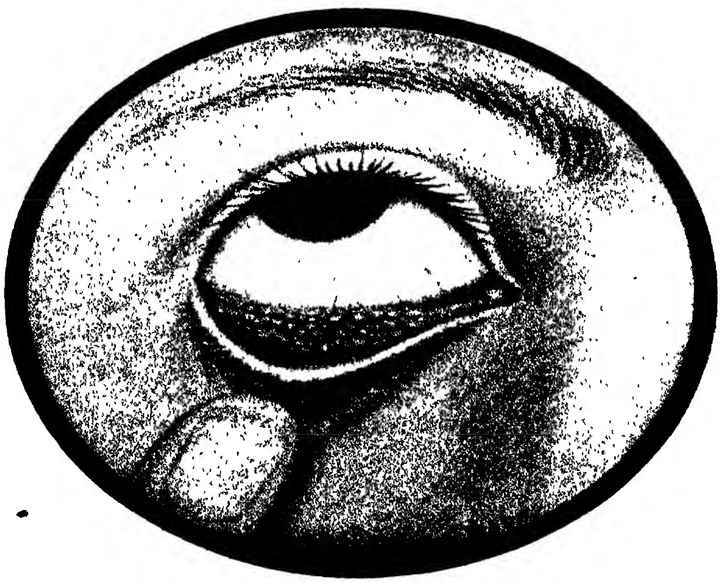


FIG. 2.—Follicular conjunctivitis.

The Macula lutea is situated about 3 mm. or 2 disc-diameters (2 p.d.) to the temporal side of the edge of the disc, and **is a little below the level of the horizontal meridian.** It is very difficult to see without a mydriatic, for the bright light on this most sensitive spot causes maximal constriction of the pupil: the corneal reflex then usually obliterates all view. It may generally be seen by using very dim illumination.

The macula varies in appearance according to illumination, refraction, complexion, &c. In general, it is a small circular area of a deeper red than the surrounding fundus, sometimes looking almost black. There is nearly always a *foveal reflex*, due to reflection of light from the walls of the foveal depression. This is most frequently seen as a silvery ring of light hiding everything behind it: it may be circular or oval, according to the incidence of the light and the refraction of the eye. Often there is an intensely bright spot at or close to the fovea, also due to reflection. The deeper red of the macula is due to the thinness of the retina, so that the specially vascular choriocapillaris of this region is seen more clearly: shadows thrown by the edges of the foveal depression may contribute a share to the deepening of the colour.

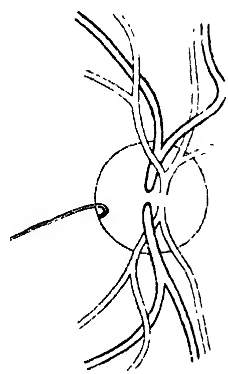


FIG. 91.—A cilio-retinal artery.

The macular region is supplied by twigs from the superior and inferior temporal arteries, and by small branches coming straight from the disc. There are no retinal blood vessels actually at the fovea (Fig. 5), and none can be seen ophthalmoscopically for a little distance around. Occasionally there are small arteries (cilio-retinal) derived from the ciliary system. They start near the edge of the disc, run inwards, and then bend sharply outwards towards the macula (Fig. 91).

The General Fundus. The appearance of the general fundus varies enormously within healthy limits. It is especially determined by the complexion of the patient, which may be taken as an index of pigmentation in different parts of the body. In people who are neither very dark nor very light the spaces between the retinal vessels show a uniform redness, occasionally with a very delicate punctate stippling, especially

towards the periphery. In albinos the choroidal vessels are seen clearly, the spaces between them being white, due to the sclerotic shining through. In partial albinism the macular region usually shows a uniform normal redness, the lack of pigmentation being manifested peripherally. Subjects of this condition are generally found to have had very light hair as infants. In very dark people the fundus is a darker red, and indications of the choroidal vessels are often seen as indefinite brighter red streaks. Sometimes the pigment between the choroidal vessels is particularly dense, or the pigment is deficient in the retinal pigment epithelium, while the choroid is deeply pigmented: the choroidal vessels are then seen separated by deeply pigmented polygonal areas (*tigroid* or *tesselated fundus*) (Plate III., Fig. 2).

There is no difficulty in distinguishing the choroidal from the retinal vessels when both are visible (*cf.* Plate X., Fig. 2). The former are broader and ribbon-like, without any central reflex streak: they anastomose freely, unlike the retinal vessels, which do not anastomose at all. Moreover, in certain parts, their anatomical distribution is very characteristic (*vide* p. 11).

All the details of the fundus will be seen much better by the direct method, unless the eye is very myopic, when the magnification is so great and the area seen is so small that it is difficult to find any particular spot.

Under normal conditions no pulsation can be seen in the retinal arteries. The retinal veins, however, may often be seen to pulsate at or near the edge of the disc, or indeed wherever they take a very sharp bend. This is usually due to transmitted pressure. The blood pressure is lowest in the veins near the disc, and there is a certain amount of obstruction to the flow of blood as the vessels pass through the narrow neck at the lamina cribrosa. With each arterial pulsation the intraocular pressure is suddenly raised slightly, so that the pressure on the outside of the walls of the veins is increased. This causes a sudden increased obstruction to the outflow of blood from the eye; the wall of the vein becomes slightly compressed, recovering itself during the arterial diastole. Hence pulsation is observed, and it will be seen best where the intravenous pressure is least, viz., nearest the heart, *i.e.*, at the disc, and where there is any additional obstruction, viz., near the lamina cribrosa and at any sharp bend. The venous pulsation can be increased or made manifest if absent by slight pressure on the globe, which has the effect of increasing the

intraocular pressure. This normal venous pulse is seen without the artificial aid of pressure on the globe in 70 to 80 per cent. of people. It will be noticed that it is diastolic ; it has therefore been called the *negative venous pulse.*

Two other forms of venous pulse occur in pathological conditions.

The *positive venous pulse* is presystolic, continuing into the systolic phase : it is due to tricuspid regurgitation, and is permitted by the normal insufficiency or absence of valves in the jugular veins. The *transmitted centripetal venous pulse* is an accentuation of the normal tendency of the pulse wave to progress through the capillaries into the veins, owing to the intraocular tension. It is due to venous congestion, with or without increased *vis a tergo.*

Visible arterial pulsation is always pathological. The blood pressure in the ophthalmic artery is only a few mm. Hg below that of the carotid in animals (*vide* p. 16). Considering the differences of blood supply it would be unwise to apply the result directly to man, but there is no doubt that the pressure in the central artery is far above the intraocular pressure. It would not be surprising, therefore, if the pulse wave were transmitted and could be seen. There are two reasons which militate against this : (1) the intraocular pressure damps the pulsation, and the increase in pressure which accompanies each pulsation is spread over the whole volume of the contents of the globe, and is transmitted to the plastic sclerotic ; (2) such pulsations as survive this damping effect are too slight to be observed in such small vessels by ordinary ophthalmoscopic examination.

Two types of arterial pulsation occur pathologically : (1) a true pulse wave, accompanied by locomotion of the vessels ; (2) an intermittent flow of blood, or pressure pulse. In the latter the arteries fill only with the heart beats, being empty between them ; it is only visible on the disc, and may be produced in a normal eye by external pressure upon the globe by a finger applied to the lid. This type of pulsation is a pure pressure phenomenon, and is caused by any considerable increase of intraocular tension with normal or lowered blood pressure, *e.g.*, in glaucoma, or by any considerable diminution of blood pressure with normal intraocular pressure, *e.g.*, in syncope, orbital tumours, &c. The true arterial pulse occurs in cases of aortic regurgitation or aneurysm, in exophthalmic goitre, &c. ; it is not confined to the

disc. It is equally a pressure phenomenon, but the differences of pressure are smaller.

Capillary pulsation is seen only in aortic regurgitation as a systolic reddening and diastolic paling of the disc.

The order of examination of the details of the fundus should be systematic. Applying the indirect method we obtain a general view. The patient is instructed to fix the gaze in such a direction that the disc is brought into view. It will occupy about the centre of the field, and a considerable area around will be visible. Any gross abnormality is detected at once. The shape and colour of the disc, the arrangement of the vessels, the colour of the choroidal reflex (its uniformity or tesselation), gross abnormalities (white or pigmented spots, &c.), are readily noted. The patient is then directed to look up to the ceiling, to the right, to the left, and down to the ground; in the latter position the upper lid is gently raised by a finger of the hand which is holding the large lens, as otherwise it will cover the pupillary area. In this manner the periphery of the fundus is brought into view. Even when the central parts of the fundus are uniformly tinted the periphery often displays traces of the choroidal vessels, associated with greater pigmentary stippling or a diminution of pigment. Only minute investigation with the direct method can show whether this is normal or pathological. The characteristic type and distribution of the pigmentation of retinitis pigmentosa is best demonstrated in this manner.

It cannot be too strongly emphasised that examination by the indirect method should not be neglected. The topographical distribution of lesions in the fundus is often more important for diagnostic purposes than minute details. The introduction of self-luminous ophthalmoscopes has led to some carelessness in this respect.

Having thus obtained a good general idea, the systematic examination is repeated by the direct method, paying special attention to points which the indirect method has left uncertain. The details of the disc—physiological or pathological cupping, blurring of the edges or swelling, abnormalities of the edges in the form of crescents and so on—are inspected. Attention is then turned to the vessels. Abnormalities in arrangement or distribution, the presence of cilio-retinal vessels, &c., are noted. The details of the individual vessels—their relative size, irregularity of contour or varicosity, visibility of the walls as shown by the presence of white lines along

the edges, abnormalities of the reflex streak, &c. - are investigated. The vessels are traced towards the periphery and the smaller vessels inspected. Changes often occur near the vessels, such as small hæmorrhages, white spots of exudate, &c.; these are carefully looked for.

Next the macula is examined : this should never be omitted. It may be brought into view by telling the patient to look into the light : with unintelligent patients it is best to say nothing, but fix the temporal edge of the disc and pass horizontally outwards for a distance of about two papilla diameters (a convenient unit in ophthalmoscopic topography), when the macula will be found. If the patient is not under a mydriatic or the pupil movements are not abolished by disease (optic atrophy, &c.) the light should be lowered so that the constriction of the pupil may be reduced to a minimum. The corneal reflex is always troublesome, but has to be dodged. Any abnormality at or near the macula is of the utmost importance. Black or white spots are often very difficult to distinguish from shadows or reflexes. if either can be made out by any means (*e.g.*, the use of a concave lens) to have a definite sharp contour, and if they do not seem to shift in the slightest degree when a minute movement is made with the mirror, it may be concluded that they are pathological entities.

Finally, the periphery of the fundus is investigated. It is important to know how far out we can see by the direct method. With modern ophthalmoscopes and full dilatation of the pupil it is possible to see almost to the ora serrata, especially if the sclera is slightly indented with a squint hook. The periphery, even in an emmetropic eye, is usually best seen with a low convex lens, owing to the obliquity of the axis of the rays as they pass through the crystalline lens.

point if it is 60 metres from the eye. Those in the second line will subtend 5 minutes if they are 36 metres from the eye ; those of the consecutive lines 24 metres, 18 metres, 12 metres, 9 metres, and 6 metres. Sometimes smaller letters corresponding to 5 metres, 4 metres, 3·5 metres are used.

A person with average acuity of vision ought therefore to be able to read the top letter at 60 metres, the second line at 36 metres, the third at 24 metres, and so on. Now, it would be very inconvenient to have to alter the distance between the patient and the letters to this large extent. A numerical convention which gives a fair comparative estimate of the value of different acuities of vision has therefore been adopted.

The patient is kept at a fixed distance from the types. This distance should never be less than 5 metres, or preferably 6 metres. At such a distance the divergence of the rays in the small bundle which enters the pupil is so slight that it

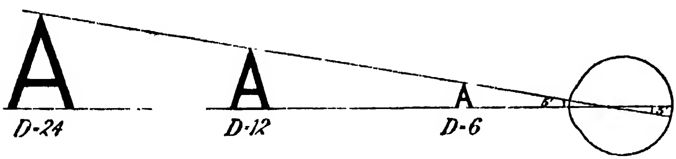


FIG. 93.

can be neglected, the rays being considered parallel. If the distance were 3 metres, for example, an appreciable amount of accommodation would have to be exerted by an emmetropic eye in order to bring the rays to a focus upon the retina : hence the estimate of distant vision would be fallacious.

A normal patient 6 metres from the types ought to be able to read every letter from the top to the end of the 6 metre line ; many people can read more in a good light. Suppose the patient can only read the 18 metre line. His distant vision is obviously defective. The numerical convention which is used to record this is a fraction in which the numerator is the distance at which he is from the types, and the denominator is the distance at which a person with normal vision ought to be able to read the last line which he succeeds in reading. The patient under consideration will therefore have his distant vision recorded thus : $V = \frac{6}{18}$. The normal patient's vision will be $V = \frac{6}{6}$

These fractions give an indication that the normal patient's vision is unity, whilst the other patient's is one-third as good. The fraction should not be reduced in this manner, because it is only an accurate numerical estimate under special conditions. It should be used merely as a convention, just as numbers are used to indicate variations in tension. If the fraction is reduced much valuable information is lost. In its original form it indicates the actual types used and the actual distance away from the types; it therefore eliminates doubt as to the accuracy of the application of the test.

The amount of illumination on the test card has a considerable influence on normal visual acuity. It has been found that the acuity rises rapidly as the illumination is increased from zero up to 5—10 ft. candles; and more slowly up to 1,000 or more ft. cs. (*v.* p. 684). The illumination of the test card should never be allowed to fall below 10 ft. candles, and it would be advantageous if the standard illumination recommended by the Council of British Ophthalmologists were universally adopted.

If the patient cannot read the largest letter he is told to walk slowly towards the types. At a certain distance he may be able to see the top letter. He should then be moved back a little, since he may not have understood exactly where to look. In this manner the farthest point at which he can distinguish the top letter is determined. If this is 3 metres, the vision is recorded thus—V = 3/60. Perhaps he is unable to see the top letter even close to it. In that case he is asked to count the extended fingers of the surgeon's hand, held up at about 1 metre against a dark background: the distance is varied to obtain about the maximum. This is recorded thus—V = fingers at 1 metre. If he cannot count fingers he is told to look at the light, either artificial or the window; the surgeon's hand is then moved between the eye and the light. If he can distinguish the movements of the hand it is recorded as V = hand movements. If he is unable to distinguish hand movements he is taken into the dark room and the light is alternately switched on and off, or light is concentrated on his eye with a convex lens or with the ophthalmoscope mirror, and he is asked to say when the light is on the eye and when it is off. If he succeeds in doing this, V = p. l. (perception of light). If he fails to see the light at all the vision is recorded as V = no p. l.

It does not follow that a patient who reads 6/6 is emmetropic: he cannot be myopic unless he is screwing up his

eyes, and in any case he cannot be very myopic. He may, however, be hypermetropic, for by an effort of accommodation he can bring the practically parallel rays emitted by the letters to a focus on his retina. We wish, then, to find out if he is accommodating. It is done by finding out the highest convex lens, placed before his eye in a testing spectacle frame, with which he can still read 6/6.

Directly a convex lens is placed before the eye in a patient who has good accommodative power it tends to make objects look blurred. Hence it is best to start with a convex lens which will quite definitely blur the types, and then gradually counteract it by placing concave lenses of increasing strength in contact with it. A + 4 D lens is put in front of the eye. Probably the patient now reads only a few lines. With the + 4 D still in position a — 0·5 D is put in front of it; the patient perhaps reads another line. The — 0·5 D is replaced by a — 1 D; he still fails to read 6/6. The — 1 D is replaced by — 1·5 D; we will suppose that he now reads 6/6. This proves that he has average normal vision with a + 2·5 D lens. With the assistance, therefore, of a 2·5 D convex lens he can relax his accommodation the corresponding amount. It by no means follows that this represents the total amount of his hypermetropia. As has already been pointed out (*vide* p. 51), the younger the person the greater the capacity to accommodate. Young people, therefore, have great facility in accommodating. If they are hypermetropic this fund has been drawn upon for a long period, so that a condition of partial contraction of the ciliary muscle becomes normal to them. Sometimes even there is excessive contraction of the muscle, so that they become artificially myopic, a condition which is called "spasm of accommodation"; it is probably diagnosed more often than it occurs.

The younger the patient, therefore, the easier it will be for him to accommodate, and the more difficult to relax his accommodation completely. That part which he can relax when convex lenses are used as described above is called his *manifest hypermetropia* (Hm.). The part which he is unable to relax, which can only be determined by paralysing the ciliary muscle, is called his *latent hypermetropia* (Hl.). The sum of the manifest and latent hypermetropia is called the *total hypermetropia*. In extreme youth nearly all the hypermetropia is latent: the lens is so resilient that it is impossible to prevent it responding to the slightest stimulus. As the lens becomes less plastic more and more of the hypermetropia becomes manifest, until,

finally, when accommodation disappears entirely, all the hypermetropia is manifest. The older the patient, therefore, the nearer the manifest hypermetropia represents the total amount.

The vision of the patient in the above example is usually recorded thus: $V = \frac{6}{6}$, Hm. + 2·5.

With intelligent patients who do their best to read as many letters as possible without continual encouragement from the surgeon, the manifest hypermetropia is obtained with less trouble by simply putting up convex lenses of gradually increasing strength until the last line which can be read becomes blurred.

An older patient than the one considered in the previous example will very likely read more with a convex glass than without it. Thus a patient of fifty-five may perhaps read only 6/12, while with a + 2 D he reads 6/6. * This man has a manifest hypermetropia of 2 D. Since he is fifty-five years of age he has only 1 D of accommodation left (*vide* p. 54). When he reads with the unaided eye he uses up this 1 D in getting as far as 6/12; he is unable to accommodate any more, so the lower letters are too blurred to read. He may manage to read 6/6 with the assistance of a + 1 D, since this, with the remnant of his accommodation, will fully correct his hypermetropia. On seeking the highest convex glass we find he can read 6/6 as well or better with + 2 D. This, therefore, represents his manifest hypermetropia. Such a case is recorded thus: $V = \frac{6}{12}$, Hm. + 2 = $\frac{6}{6}$.

* Apart from counteracting a convex lens as described above the student should not use concave lenses in testing the distant vision, unless the patient is under a mydriatic. An emmetrope, or even a hypermetrope, if neither is presbyopic, will read 6/6 quite well with weak concave lenses in front of the eye; he simply accommodates the amount which is requisite to counteract the lens. Hence, unless the patient is under the influence of a mydriatic, we learn nothing from the procedure; it is therefore redundant.

Having tested the distant vision and determined the amount, if any, of the manifest hypermetropia, the near vision should next be tested. For this purpose test types for near vision are used (Fig. 94). Snellen's are constructed on exactly the same principle as the distant ones, and are therefore theore-

tically more accurate. The legibility of small types has been found empirically to be increased by slight modifications in the breadths and forms of the letters (*vide* p. 687). Ordinary types in common use are therefore more legible than Snellen's types of corresponding size. Jaeger's near test types, which are very frequently used, are simply the ordinary printer's founts of type, from the smallest upwards (nonpareil, minion, &c.). They are sufficiently accurate for practical purposes.

The patient is told to hold the test card. The position where he holds it of his own accord will often impart useful information. If he is old and holds it a long distance away, he is most likely to be an emmetropic or hypermetropic presbyope. If he holds it closer than the ordinary reading distance and reads the smallest type fluently, he is probably myopic, whatever his age may be, though children often get into the habit of holding books unnecessarily close.

Take first the example of an emmetrope. We find that he reads 6/6, that he has no manifest hypermetropia, and that he reads Jaeger 1, holding it of his own accord at ordinary reading distance (22 cm. or 9″). This is recorded—V = 6/6, no Hm., J. 1. If no distance is stated in recording the near vision it is understood to be the normal distance.

Take now a patient who reads only 6/60, has no manifest hypermetropia, but reads Jaeger 1 fluently, only, however, when the card is held closer than normal to the eye. In this case the distance at which the card is held should be guessed or measured. Let us suppose that it is about 5 inches. This is recorded—V = 6/60, no Hm., J. 1 at 5″.

Take now a patient of fifty-five who reads 6/6, and has no manifest hypermetropia. We give him the near types, and he holds the card a long distance off, but even so cannot read Jaeger 1. In this case it is waste of time to discover exactly which type he can read, and to measure the distance at which he can read it. We know that as he is fifty-five he has only 1 D of accommodation remaining (*vide* p. 54). What we wish to know is whether he can read Jaeger 1 at ordinary reading distance if we correct his presbyopic defect. We therefore at once put up a + 3 D lens before the eye, tell him to hold the types closer, and ask him if he can read the smallest. He will probably do so easily. This is recorded—V = 6/6, no Hm., $\bar{c}$ + 3 = J. 1.

Finally, take a patient of fifty who reads 6/12, but with + 2 D reads 6/6. He will hold the near types a long distance away as in the last example: if we investigate the question he

J. 1 (Sn. 0.5). 50 cm.

to the W S W at a very great distance by my guess, it could not be less than fifteen or twenty leagues off I saw abundance of parrots, and fain I would have caught one, if possible, to have kept it to tame, and taught it to speak to me I did, after some painstaking, catch a young parrot, for I knocked it down with a stick, and having recovered it, I brought it home; but it was some years before I could make him speak However, at last I taught him to call me by my name very familiarly

J. 2 (Sn. 0.6). 60 cm.

But the accident that followed, though it be a trifle, will be very diverting in its place. I was exceedingly diverted with this journey. I found in the low grounds hares, as I thought them to be, and foxes; but they differed greatly from all the other kinds I had met with, nor could I satisfy myself to eat them, though I killed several.

J. 4 (Sn. 0.8). 80 cm.

with a row of stakes, set upright in the ground, either from one tree to another, or so as no wild creature could come at me without waking me. As soon as I came to the seashore, I was surprised to see that I had taken up my lot on the worst side of the

J. 6 (Sn. 1). 1 m.

and from home. However I travelled along the shore of the sea towards the east, I suppose about twelve miles, and then setting up a great pole upon the shore for a mark, I concluded I would go home again, and that the next journey I took should be on the other side of the island,

J. 8 (Sn. 1.25). 1.25 m.

east from my dwelling, and so round till I came to my post again; of which in its place. I took another way to come back than that I went, thinking I could easily keep all the

J. 10 (Sn. 1.5). 1.5 m.

gun, ammunition, hatchet, and other things very heavy. In this journey my dog surprised a young kid, and seized upon it, and I running in to take

J. 12 (Sn. 1.75). 1.75 m.

for I was very impatient to be at home, from whence I had been absent above a month. I cannot express what satisfaction it was to me to

J. 14 (Sn. 2.25). 2.25 m.

Come into my old hutch, and lie down in my hammock bed. This little wandering journey,

Fig. 94.—Test-Types for Near Vision.

Jaeger Test-Types, with approximate Snellen equivalents, and the most remote distances at which each should be read with average normal vision.

will not be able to read nearly as well even a long distance away as the last patient. He has only 2 D of his accommodation remaining, but he also has 2 D of hypermetropia. We cannot therefore expect him to read Jaeger 1 at ordinary reading distance unless we not only correct his presbyopia but also his hypermetropia. We therefore at once put up a + 4 D, and find that he reads Jaeger 1 at ordinary distance quite well. This is recorded—V = 6/12, Hm. + 2 = 6/6, $\bar{c}$ + 4 = J. 1.

The ordinary rule of presbyopic loss of accommodation, viz., 1 D for each five years after forty, is a liberal allowance, and we often find that patients are more comfortable with less (*vide* p. 55). The lower correction should be ordered and only in very exceptional cases should more be ordered.

An indication of the *range of accommodation* is given by the knowledge of the manifest hypermetropia, combined with the ability to read the small types at ordinary reading distance. Strictly, the accommodation should be more carefully tested in each case, but this is often neglected.

The method adopted to find the near point of the eye has already been mentioned (*vide* p. 50). For practical purposes it is sufficient to use the smallest Jaeger or Snellen near type and approach it nearer and nearer to the eye until it can no longer be read. The last point at which it can be read gives the near point. The distance of the near point from the eye is then measured with a tape. This distance is transformed, if necessary, into millimetres (25 mm. = 1 inch), and the range of accommodation is deduced from the formula A = P—R (*vide* p. 51). Of course, the full range of accommodation in a hypermetrope cannot be accurately arrived at unless the total hypermetropia is known; this may require the use of a mydriatic. Practically, however, we are chiefly concerned in discovering paralysis or paresis of accommodation, such as may occur after diphtheria or previous use of a mydriatic. In these cases the knowledge of the distance of the near point is sufficient.

The next step—one which is far too often neglected—is in every case to test the *pupil reactions* and record them. If the visual tests have shown deficiency it may be necessary to use a mydriatic, in which case it will be impossible to test the pupil reactions at a later stage of the same visit; hence the importance of recording them at once.

We should also test the field of vision roughly.

The Field of Vision.—There are several methods of testing the field of vision.

(1) A rough, but very useful, method, the so-called confrontation test, which should be applied in every case, at any rate if there is the slightest suspicion of defect, is as follows :—

The surgeon stands facing the patient at a distance of about 2 feet. The patient covers his left eye with the palm of his hand. He is told to look straight into the surgeon's left eye. The surgeon closes his right eye. He then moves his hand in from the periphery towards the common line of vision of the patient's right and his own left eye, keeping his hand in the plane half-way between the patient and himself. Directly he sees it himself the patient ought to say that he also sees it. The movements of the hand are repeated in various parts of the field—above, below, to the right, to the left, and so on.

This method is extremely simple, rapidly applied, and an excellent test. It will be seen that the surgeon tests the range of the patient's field by that of his own, which may be considered normal; moreover, he is continually watching the patient's eye, so that he can at once observe any deflection from the point of fixation.

The gross defects in the field which are most likely to escape recognition are homonymous and bitemporal hemianopia, the latter usually due to acromegaly or tumour of the pituitary body (Chap. XIX.). They may be roughly tested for by telling the patient to look straight at the surgeon, situated as before, both eyes being open. The surgeon holds up both hands, one in each temporal field, and the patient is told to touch the surgeon's hand. If he asks "Which one?" he has not bitemporal hemianopia, since he sees both hands. If he promptly points to one hand he should be asked if he sees the other; if he does not, he probably has homonymous hemianopia.

If any defect is indicated by these methods or is suspected from other features of the case it must be accurately mapped out and recorded with the perimeter.

(2) *The Perimeter.*—The perimeter consists essentially of an arc, marked on the back in degrees of a circle, capable of being revolved round a pivot which the patient fixes with the eye under examination (Fig. 95). The chart, which has concentric circles marked upon it, corresponding with the degrees on the arc, is under the surgeon's control at the back of the perimeter. In self-registering perimeters, which are almost invariably used, the readings are recorded by perforations with a sharp point.

The details of taking a perimetric chart can only be taught by actual demonstration. It will suffice to emphasise here the chief procedures to be followed in order that accuracy may be attained.

The patient is seated with his back to the light. His chin

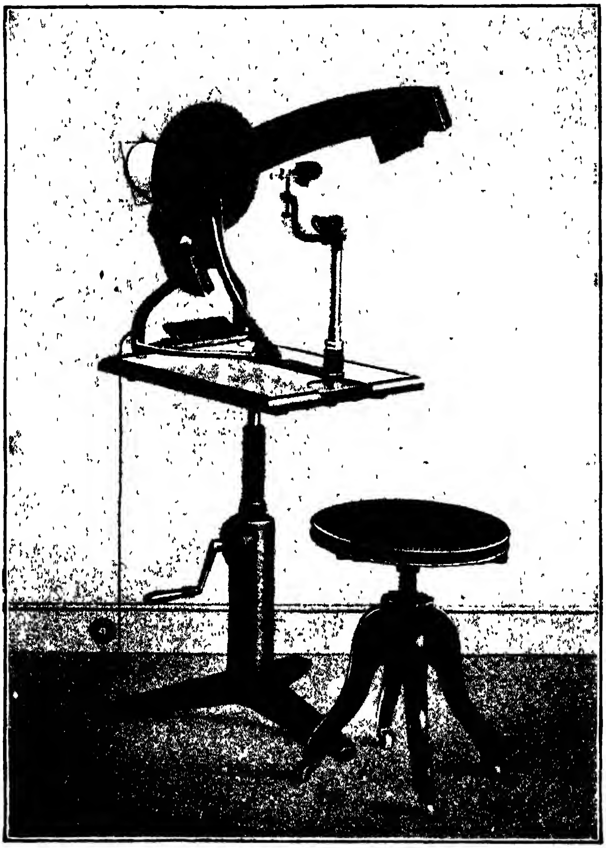


FIG. 95.—Lister perimeter and scotometer.

rests upon the chin-rest; the face is vertical and not tilted to one side; one eye is covered. The other eye, situated at the centre of the arc, fixes the white dot around which the arc revolves.

The field should first be taken with a white object 10 mm. in diameter. At least eight meridians must be investigated,

preferably sixteen. If the field is very small a 5 mm. square should be substituted, and the process repeated. In each meridian the object should be carried quite up to the fixation point, as there may be areas inside the limits of the field which are blind (*absolute scotomata*). These should be mapped out with the same accuracy as the extreme limits of the field. If the scotomata are small the limits may be determined with an intelligent patient best with a very small object, *e.g.*, 2 mm. square. The size of the test object and the distance from the perimeter may be conveniently recorded by a convention similar to the mode of recording visual acuity, *e.g.*, 10/300, both distances being expressed in millimetres (A. H. H. Sinclair). With small objects *relative scotomata* can be found which are not demonstrable with large objects.

Having mapped out the field for white the process should be repeated with similar, but coloured, objects. The limit of the field for a colour is the point at which, passing from the periphery to the centre, the colour first becomes evident. Peripheral to this limit the object may still be perceptible but appears grey. The exact limit is difficult to determine, for most colours change in hue and saturation as the object passes from the fixation point towards the periphery. Red or green should be used first, then blue or yellow. Under ordinary conditions, the blue field is largest, slightly smaller than the white: then follow the yellow, red and green, in the order named (Fig. 96). There is a particular purplish-red and a particular bluish-green (490 $\mu\mu$) which have the same field; and similarly a particular blue (460 $\mu\mu$) and a particular yellow (570 $\mu\mu$). These pairs of colours are complementary, *i.e.*, a mixture of the red and the green, or of the blue and the yellow, produces white.

The extent of the normal field, with a 10 mm. square, under good illumination, is shown in the accompanying chart (Fig. 96). The peculiar shape is essentially due to the shape of the sensitive area of the retina as projected outwards, but is often modified when the field is taken in the ordinary manner by interference caused by the nose and the brows; this complication can be eliminated if, when the field of the right eye is being taken, the head is turned somewhat to the left, and *vice versâ*. It is seen that the field for white extends upwards 45°, outwards rather more than 90°, downwards 70°, and inwards 60°. The size varies with the illumination, the size of the test object, the contrast of the test object with the background, and the state of adaptation of the eye. The field for

blue and yellow is roughly 10° less in each direction, that for red and green another 10° less. The limits of the colour fields vary not only with the intensity of the light, but also with the saturation of the colour, and above all with the size of the object. If these are sufficiently great, colours may be recognised almost, if not quite, at the periphery.

Even the ordinary perimetric observation is a relatively rough test and purely subjective. Every student should have

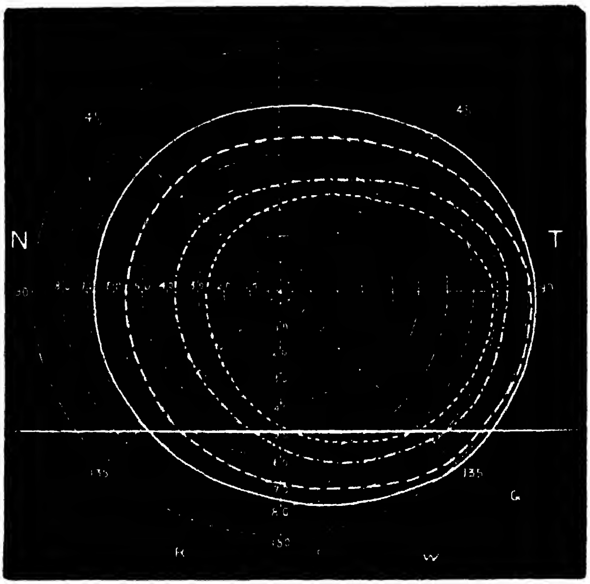


FIG. 96.—Perimeter chart of right eye (Landolt). T, temporal side; N, nasal side; W, for white object; B, for blue; R, for red; G, for green.

his own field taken: he will then appreciate the difficulties which patients experience. The normal physiological response to an object in the peripheral field is to turn the eyes towards it. In charting the field of vision this normal response has to be suppressed, fixation being rigidly maintained while attention is directed to an object at the periphery. Hence the first fields taken must always be regarded with suspicion, and particularly so in the case of dull or neurotic patients. The most variable factor is the illumination, and sufficient attention is not usually paid to this point. With good illumination an object subtend-

ing a visual angle of 0·5° will give the full formal field for white. The ordinary 10 mm. object at the distance generally used, viz., 30 cm., *i.e.*, 10/300, corresponds with a visual angle of 2°. Deductions made from variations in the colour fields are particularly unreliable (*vide* p. 141).

Special care must be taken to investigate the central part of the field for red and green, since conditions are not uncommon, *e.g.*, tobacco amblyopia and retrobulbar neuritis, in which these colours are not recognised by central vision (*central relative scotomata*). The 5 mm. square should be placed over the point of fixation and the colour changed;

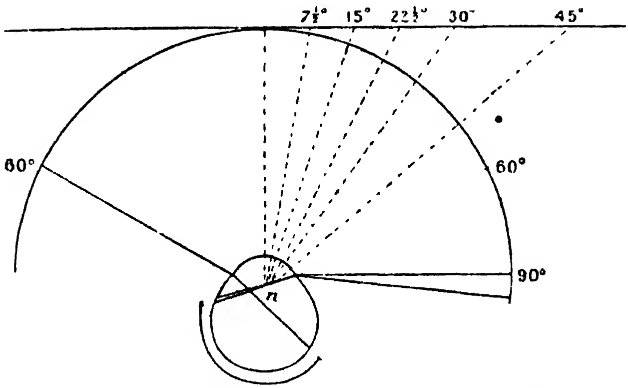


FIG. 97.—Diagram of the right eye, showing the relationship of the retina to the degrees of the perimetric arc, and the relative value of the latter when projected on a tangent scale. *n*, nodal point. F, point of fixation.

blue and yellow will be recognised as such, but not red and green.

(3) For more accurate investigation of details another method must be employed, but it is applicable only to the central and paracentral areas. It consists in placing the patient 2 metres from the centre of a large black screen, 2 metres or more in diameter (Bjerrum's screen). The patient fixes a spot in the centre of the screen and smaller circular discs of ivory, 1 mm. to 10 mm. in diameter, attached to a long black rod are brought in from the periphery on a level with the screen. At this distance a 3 mm. object subtends a visual angle of about 5 minutes. It will be noticed that, the angles being projected on to a flat surface, tangents are recorded, not angles themselves as with the arc. Hence only a small area

can be investigated, and the distortion must be taken into account. Some points of diagnostic importance which cannot be elicited by the perimeter can be brought out by this method. Various *scotometers* have been devised on the principle of Bjerrum's screen.

If the charts of the two eyes are superposed there will be a large central area which is common to both eyes: this is the *field of binocular vision.*

The Light Sense may be roughly tested by means of Bjerrum's test types, which consist of Snellen's test types printed grey on a grey background of different intensity; or it may be tested against the surgeon's as a control by using the ordinary Snellen's test types and gradually lowering the illumination. There are considerable individual differences in the rate of development of dark adaptation and facility of behaviour under low illumination. These are of great importance in night-operations by the combatant services and to civilians during "black out." Special methods have been devised for their investigation. The administration of vitamin A, which is essential to the formation of visual purple, does not seem to improve scotopic vision unless it is deficient in the diet. The rate of dark adaptation may be much prolonged in pathological conditions, *e.g.*, retinitis pigmentosa, xerophthalmia, glaucoma, &c.

The Colour Sense requires elaborate apparatus for its scientific investigation. The methods used will be discussed later (p. 418).

SECTION III

DISEASES OF THE EYE

CHAPTER X

Diseases of the Conjunctiva

THE conjunctiva shows very considerable variations in appearance at different ages and in people who follow various employments. The peculiarities of colour, vascularity, laxity, &c., which are consistent with health can be learnt only by repeated observation.

It is necessary for a scientific appreciation of pathological conditions to be cognisant of the normal structure of a part. The conjunctiva is divided into two portions, palpebral and bulbar; the folds uniting these parts are the fornices. The palpebral conjunctiva is said to commence at the anterior margin of the edge of the lid, but from this point to the posterior margin of the edge (the intermarginal strip) and for about 2 mm. beyond (to the sulcus subtarsalis) there is a transitional zone covered with stratified epithelium and partaking of the characters of both skin and conjunctiva (Chap. XXXI.). There are two layers of epithelium over the palpebral conjunctiva: from the fornices to the limbus the epithelium becomes gradually thicker, forming eventually again a stratified epithelium. Below the epithelium is an adenoid layer, consisting of loose connective tissue containing mononuclear lymphocytes: below this a fibrous layer, much denser and passing insensibly into the underlying tissues—lid or sclerotic. The palpebral conjunctiva is firmly adherent to the tarsus, while the bulbar portion is freely movable over the sclerotic except close to the cornea.

Bacteriology. The conjunctival sac is practically never free from organisms. Owing to the relatively low temperature of the conjunctival sac due to exposure, evaporation of lacrymal fluid and moderate blood supply, bacteria do not propagate themselves readily. The tears are not a good culture medium, and though they contain lysozyme, they cannot be regarded as actively bactericidal. They contain no agglutinin, and diphtheria antitoxin does not

pass into them when present in the blood. Hence they act principally in a mechanical manner, washing away deleterious agents and their products. The presence of dust, even if it be sterilised, augments the bacterial content of the conjunctival sac. It is also increased by bandaging owing to the arrest of movement of the lids and the raised temperature of the sac. Most of the organisms normally present are non-pathogenic, but some of these are morphologically identical with pathogenic organisms. Diplococci indistinguishable from pneumococci are found; they may be innocuous to animals or prove themselves true pneumococci. The conjunctiva of many people is immune to pneumococcic infection, though the same germs transferred to other persons will excite a violent inflammation. It may be stated at once that the pneumococcus is one of the most dangerous organisms in the pathology of the eye. Another bacterium, the so-called xerosis bacillus, is morphologically identical with the diphtheria bacillus; it can only be distinguished by skilled examination of cultures. Staphylococci are found; they are relatively innocuous in the absence of other organisms, but play an important part in mixed infections. Staphyloccoccus albus and xerosis bacilli are frequently present in the normal conjunctival sac. There are reasons for thinking that their presence favours the growth of pathogenic bacteria: thus xerosis bacilli promote the multiplication of Koch-Weeks' bacilli both in cultures and on the conjunctiva. Streptococci, Bac. coli, &c., are pathogenic, but rare. Other pathogenic organisms—gonococci, Koch-Weeks' bacilli, diplobacilli—will be discussed later.

Inflammation of the Conjunctiva

Conjunctivitis. Inflammation of the conjunctiva manifests itself in many grades and many types. It is always accompanied by hyperæmia and by increased secretion. The hyperæmia varies in degree and in distribution: the secretion varies in nature and amount.

Hyperæmia may be transitory, or recurrent and chronic. The former is caused by temporary irritation, as by a foreign body in the conjunctival sac (which includes the surface of the cornea), concretions in the palpebral conjunctiva (*vide* p. 189), in-growing lashes (*vide* p. 620), &c.: in such a case the increased secretion is almost wholly a reflex secretion of tears. A foreign body, especially a grain of corn or the wing capsule of an insect, may be retained in the fornix and set up a violent unilateral conjunctivitis. Occlusion of the canaliculus

(*vide* p. 652) is easily overlooked. Irritation limited to the lower fornix may be artificial in malingerers and hysterical patients.

Recurrent or chronic congestion may be caused by the conditions of life—dusty, ill-ventilated rooms, exposure to strong light, &c. Bright light, especially exposure to tropical sunlight, acts partly by the glare due to the luminous rays, partly by the chemical action of the actinic, especially the ultra-violet rays, and partly by the heat, which is chiefly due to the infra-red rays. Chronic congestion is often due to conditions remote from the conjunctiva itself. Very frequently it is a reflex irritation due to errors of refraction; in such cases the edges of the lids may participate. Other causes are found in errors of metabolism—gout, over-eating and drinking, and so on. It is a characteristic symptom of hay fever, and in this case there may be an excess of eosinophile leucocytes in the conjunctival secretion.

Simple hyperæmia of the type described causes a sense of discomfort, often described as tightness, grittiness, inability to keep the eyes open, tiredness, &c. Bright light is resented, but there is seldom true photophobia. The conjunctiva often looks quite normal until the lower fornix is exposed, when it will be seen that the parts in contact are congested and sticky. The discomfort frequently comes on only in the evening or after near work. In the gouty cases there may be œdema—*chemosis*.

Chemosis affects the most loosely attached parts of the conjunctiva, *i.e.*, principally the bulbar conjunctiva and fornices. The mucous membrane becomes swollen and gelatinous in appearance. The swollen membrane forms a wall around the cornea, which it may overhang in severe cases. The palpebral conjunctiva is little affected, but the tissues of the lid are often also œdematous, so that the lids are swollen and the upper hangs down over the lower.

Whenever watering of the eyes is complained of, and whenever only one eye is congested or shows signs of conjunctivitis, the lacrymal passages must be investigated. Pressure with the finger backwards and inwards over the lacrymal sac may cause regurgitation of fluid—tears, mucus, or pus—showing that the outflow into the nose is obstructed. If no regurgitation can be detected, the position of the lower punctum must be noted. It ought to be invisible until the lid is slightly everted.

The *treatment* of simple hyperæmia consists primarily in the removal of the cause. Defective conditions of life must be ameliorated if possible. The irritation of strong light must

be removed, or modified by the use of dark glasses. If the light is not very excessive ordinary neutral tinted ("smoked ") glasses will suffice. They are better than blue or other coloured glasses as they reduce the intensity of the luminous rays more uniformly throughout the spectrum. Nearly all kinds of glasses cut off a large percentage of the ultra-violet rays, especially those of shortest wave length. Sir William Crookes prepared a series of synthetic glasses which have various absorptive powers. Some of these cut off practically all the infra-red and ultra-violet rays, while absorbing the luminous rays to only a slight degree. Tinted Crookes's glasses are specially indicated for use in tropical climates and for winter sports.

Errors of refraction must be corrected. It should be remembered that the error may be artificial, through the use of wrong spectacles. The amount and conditions of near work should be specifically stated.

Defects of the lacrymal apparatus must be treated (Chap. XXXII.). If no defect is noted, local treatment of the hyperæmia is ordered for a time; but if the condition does not improve, the patency of the lacrymal passages must be demonstrated by syringing. The beginner must be careful, however, that he does not do harm rather than good.

Errors of metabolism must be treated on general medical principles. Such causes are easily overlooked; hence they should be specially borne in mind.

Local treatment consists in bathing the eyes frequently with warm boric lotion, with or without a mild astringent, *e.g.*, zinc sulphate, gr. ½ or i to ℥ i. A drop of a mixture of equal parts of tincture of opium and distilled water, night and morning, will be found soothing. Hazeline, 20 minims to ℥ i., is sometimes useful, but varies in its effect in different people. Cocaine must be used with diffidence: its effects are transitory, and it has a deleterious action upon the corneal epithelium, but in quite weak doses often affords much comfort.

In cases where temporary alleviation—usually of the disfiguring signs—is insistently desired, a drop of adrenaline solution (1 in 1000) instilled into the eye will remove the discomfort and reduce the redness of the conjunctiva. The effect is, however, very transitory, but it will often earn gratitude. It is especially useful after the removal of a foreign body from the cornea.

The *nature of the secretion* in conjunctivitis is of diagnostic importance. It may be watery, mucous, muco-purulent, or

purulent, and the disease is often classified accordingly. Most forms of acute conjunctivitis are due to bacterial agency. Unfortunately, each pathogenic organism does not produce a specific clinical picture. It is therefore wise in the meantime to retain the old clinical terminology.

Watery secretion is usually due to reflex secretion of tears. The other types of secretion show some relation to the bacterial cause, and must be distinguished on account of the information they convey as to the probable severity of the condition and the indication they provide for special measures of treatment.

The chief forms of conjunctivitis may be divided into two groups: acute, and sub-acute or chronic. Acute conjunctivitis may be classified as simple acute (including muco-purulent), purulent, membranous, and phlyctenular. Sub-acute or chronic conjunctivitis includes simple chronic conjunctivitis, angular conjunctivitis, follicular conjunctivitis, trachoma, tubercle, and syphilis.

Simple Acute Conjunctivitis (*Syn.—Catarrhal Conjunctivitis*). The condition described as hyperæmia of the conjunctiva passes imperceptibly into a condition characterised by greater and more general hyperæmia and a thicker mucous discharge which gums the lids together. The lids are usually described as being stuck together in the mornings, because the condition is most noticed after they have been closed for a considerable period. The causes, symptoms, and treatment are the same as in simple hyperæmia.

Various more intense forms of simple acute conjunctivitis are met with: they are probably all of bacterial origin, the organisms differing in different cases. Among the lower classes the disease is called "blight" and it is commonly attributed to a "cold in the eye." Cold probably acts only by lowering the resistance of the tissues to the action of organisms.

The commonest form is **Muco-purulent Conjunctivitis.** Here, as the name implies, the secretion is muco-purulent; it is more profuse than in the simpler forms. As in most cases of conjunctivitis the disease is contagious, being transmitted directly by the discharge and possibly by the air of ill-ventilated rooms, though this method is doubtful, since most of the organisms are non-sporing and are easily destroyed by drying. The whole conjunctiva is a fiery red ("pink eye"); all the conjunctival vessels are congested, except the circumcorneal zone in the milder cases (*vide* p. 83). Flakes of muco-pus are

seen in the fornices, and often between and upon the margins of the lids. If the discharge is allowed to dry the lashes become matted together by dirty yellow crusts. These may be easily mistaken for the condition found in blepharitis, but, if the crusts are bathed off, the underlying lid margins will be found healthy. Flakes of mucus passing across the cornea often give rise to coloured haloes, owing to their prismatic action. These "haloes" must be carefully distinguished from those met with in glaucoma (*q.v.*).

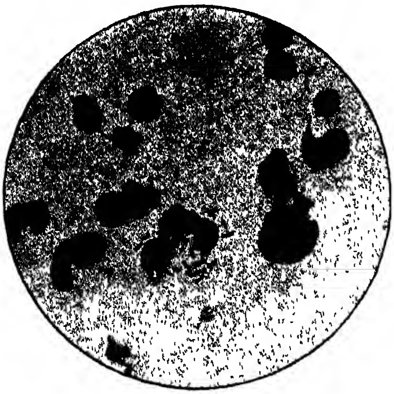


FIG. 98.—Koch-Weeks' bacilli (× 1000)

The discharge is at first mucous, but gradually becomes more purulent. Beginners are liable to mistake muco-purulent for true purulent conjunctivitis. In the former, the more purulent masses are found among the lashes and at the inner canthus, whilst the fornices and bulbar conjunctiva show only flakes of translucent or yellowish secretion. In the latter, crusts of inspissated pus may be seen among the lashes and at the canthi, but when the lids are separated fluid pus wells out.

The disease reaches its height in three or four days: if untreated it is liable to pass into a less intense, chronic condition. Complications are rare, but abrasions of the cornea are liable to become infected and to give rise to ulcers. Occasionally marginal ulcers form—in debilitated or old people, or as the result of improper treatment.

Muco-purulent conjunctivitis frequently complicates phlyctenular conjunctivitis.

Pathology. Muco-purulent conjunctivitis is frequently caused by the Koch-Weeks' bacillus (Fig. 98). This is a very slender rod, varying much in length. It stains badly with the ordinary basic dyes, *e.g.*, Löffler's methylene blue; it is decolourised by Gram. Groups of bacilli found in much degenerated "skeletonised" pus cells are very characteristic. The organism is rapidly destroyed by drying. It has been known to give rise to very definite epidemics. In England

the cases are usually sporadic, though moderate transmission is common. An attack confers immunity for some time.

The Koch-Weeks' bacillus is by no means the only cause of muco-purulent conjunctivitis. Diplococci which are indistinguishable from pneumococci (Fig. 99) also cause it, probably more frequently in England. Pneumococcic conjunctivitis, though not definitely separable from the other acute forms clinically, shows distinct tendencies which should be borne in mind, the more so since the pneumococcus is the cause of hypopyon ulcer (*q.v.*). There is usually more œdema (chemosis), small ecchymoses are common, and a membranous film may form—"pseudomembranous conjunctivitis." It is commonest in northern countries and in the cold weather, and is more often found in children than adults. It ends with a crisis, like pneumococcic infection of the lungs, after which the organism rapidly disappears from the secretion. It is often accompanied by nasal catarrh, which may precede or follow the inflammation. Iritis is very rare as a sequel of conjunctivitis, but pneumococcic conjunctivitis is exceptional in this respect. The inflammation of the iris is set up by absorption of toxins (*cf.* Hypopyon Ulcer of the Cornea).

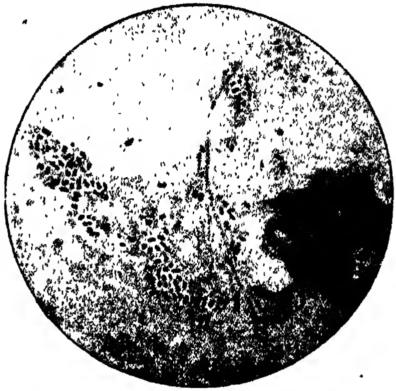


FIG. 99.—Pneumococci ($\times$ 1000).

The "influenza bacillus," which is distinguished with difficulty from the Koch-Weeks' bacillus, is responsible for conjunctivitis during influenza epidemics, more often in children than adults.

The muco-purulent conjunctivitis associated with phlyctenular conjunctivitis is usually due to staphylococcus aureus, which may also set up conjunctivitis in cases of blepharitis and eczema or impetigo of the skin. The organism sometimes causes a muco-purulent discharge after cataract extraction and other operations, and also in the new-born, probably owing to the defective resistance of the tissues in these conditions. In the presence of irritating dust a staphylococcic conjunctivitis of slight intensity may be set up. The presence of

staphylococci is liable to aggravate conjunctivitis set up by some other organism.

Other organisms have been found, but their ætiological relationship to the disease is not proved.

Muco-purulent conjunctivitis generally accompanies or follows measles and frequently scarlet fever. It also occurs with acne rosacea, when minute nodules, somewhat like phlyctens, form at the limbus and on the cornea. It frequently recurs in these cases, and is intractable. Muco-purulent conjunctivitis sometimes occurs among people who use swimming baths (*v.* p. 182). It often spreads rapidly through schools and other such institutions.

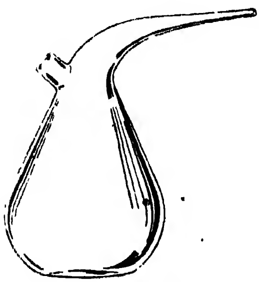


FIG. 100.—Undine.

Treatment. The treatment for muco-purulent conjunctivitis consists of two main procedures: (*a*) the frequent washing out of the conjunctival sac with a suitable lotion; and (*b*) the control of the infection by appropriate drugs. The lotion should be warmed and diluted by the addition of a little boiling water. It is well for the surgeon himself or a competent nurse to wash out the conjunctival sac the first time. For this purpose an "undine" (Fig. 100) is the most convenient reservoir. The lids are everted and the lotion is poured from a little height over the whole surface, every crevice being irrigated as thoroughly as possible. The nozzle of the undine should not be allowed to touch any part of the eye. The patient may be directed to use the ordinary eye-bath for the application of eye lotions. If a child, the parents should be instructed to hold the lids apart, the child lying upon its back. A pad of cotton-wool, dripping with the lotion, which may be warmed, is then held over the eye and the lotion squeezed out: the process is repeated until all discharge has been washed away. The lotion may be warmed by placing the bottle in hot water, but the addition of an equal part of hot water suffices. Eye lotions act chiefly by washing out deleterious material, since they cannot be used sufficiently strong to act as efficient antiseptics. Some considerable antiseptic effect, however, may be gained by using solutions such as sublimate lotion (lotio hydrargyri perchloridi 1 in 10,000), but on the whole boric acid lotion is probably nearly as efficacious and is less irritating. Indeed, boric acid lotion or

even normal saline solution should be ordered for nervous people who are afraid of a little pain, since it is useless to bathe the eyes unless the fluid irrigates the conjunctival sac.

Control of infection is most effectively maintained by the use of bacteriostatic drugs. If the organism is sensitive to it (and most conjunctival organisms are), penicillin is the most effective, administered as drops (500 to 1,500 Oxford units per c.c.) every two or three hours. It should be administered a little time after an irrigation. The value of penicillin is greatest in the acute stage: when the infection has become chronic its effect is frequently temporary and relapses are common. In these circumstances it cannot replace the older methods of treatment. An alternative but much less efficient drug, if penicillin is not available, is sodium sulphacetamide (albucid soluble) as drops in 30 per cent. solution.

Boric acid ointment, albucid ointment, or sterile vaseline is smeared along the lids at bed-time, or, in children, as often as they are put to sleep: it prevents the lids from sticking together—a two-fold benefit, that of preventing discharge from being retained, and that of obviating pain on opening them. A penicillin ointment (1,000 units per gm.) may be employed, but it is not so efficacious as the drops, and has a greater tendency to cause irritation.

The eyes should not be bandaged, as it prevents the free exit of the secretion. If there is any photophobia a shade or dark goggles should be worn. The patient should spend as much time out of doors as possible.

If this treatment is properly carried out, the patient will be well in a few days. Even if only partially successful there will then be less discharge.

If the case is not progressing as rapidly as could be desired, or the attendants are not reliable, and if the discharge is subsiding, it is wise to paint the lids once with silver nitrate solution (gr. x. to ℥ i.) This is the strength which should always be used for painting lids. Stronger solutions act too vigorously as caustics, and, if a caustic effect is desired, it can be obtained with greater precision by other means. Weaker solutions are precipitated by the chlorides in the lacrymal secretion, so that they are practically useless.

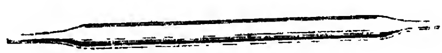


FIG. 101.—Glass rod.

The following is the best method of painting lids. A glass

rod is used, tapering at each end (Fig. 101). The finger should be passed over the ends each time before use to make sure that they are not chipped. The end is dipped in lotion to damp it. A *very thin* wisp of cotton-wool is then tightly wound round the end, starting where it begins to taper: this fixes the wool. The end of the wisp is left loose, so that it may absorb the solution. The other end of the rod is armed in the same manner.

The patient, if a child, is placed upon his back. The lids are everted; the wool, dipped in the silver solution, is applied freely to the conjunctival sac, the cornea being protected as much as possible. It is quite unnecessary to neutralise the excess of nitrate with salt solution, as is often taught. The excess may be mopped up with a pad of dry absorbent wool. If, as is usually the case, the other eye is affected, the other end of the rod is used in the same manner. In the absence of a glass rod the best implement is an ordinary wooden match, used in the same way. The match can be thrown away after use. The glass rod must be sterilised by boiling. A camel's hair brush should not be used: it cannot be kept aseptic.

A single painting with silver nitrate will often produce an excellent result. It is good as a prophylactic if discharge is inadvertently introduced into a normal eye. Other preparations of silver—protargol (20 per cent.), argyrol (25 per cent.), &c.—are not so efficacious, but they have the advantage of being less painful.

Silver nitrate acts by forming a thin epithelial eschar and coagulating the muco-purulent discharge. The bluish-white film is cast off in flakes, and until this process is complete the feeling of a foreign body in the eye is experienced. The irritation is reduced if the lid is kept everted for a few minutes and the flakes gently removed with cotton-wool, a drop of 2 per cent. cocaine solution being then instilled. Silver nitrate is not strongly bactericidal, but the organisms are entangled in the coagulum and removed with it. Moreover there is a powerful physiological response to the caustic; hyperæmia increases and the tissues are flooded with blood serum, which can thus more effectively exert its bactericidal and antitoxic powers. The slighter efficacy of protargol and the modern colloid substitutes for silver nitrate is to be attributed to their smaller caustic and irritant properties, which are often regarded as their chief advantages.

The conjunctiva generally returns to a perfectly normal condition. If the case has been neglected and chronic inflam-

matory signs persist, astringents should be used as for chronic conjunctivitis (*q.v.*).

Since the disease is contagious care must be taken to prevent its spread. The patient must keep his hands clean and no one else must be allowed to use his towel, handkerchief, &c.

Purulent Conjunctivitis (*Syn.—Acute Blennorrhœa, Gonorrhœal Conjunctivitis*) is a much more serious condition. It occurs in two forms—as ophthalmia neonatorum in babies, and as gonorrhœal conjunctivitis in the adult. Certainly the former, and probably the latter, is not invariably gonorrhœal.

Gonorrhœal Conjunctivitis (*Syn.—Acute Blennorrhœa of Adults, &c.*). Gonorrhœal conjunctivitis is even more serious in the adult than in babies; fortunately, considering the prevalence of gonorrhœa, it is comparatively rare (1 in 700—800 cases). While generally due to the gonococcus it is important from the medico-legal point of view to remember that the same features may be found with streptococci, diphtheria, and with mixed infections. The gonococcus is a bun-shaped diplococcus, staining readily, decolourised by Gram and found within both leucocytes and epithelial cells (Fig. 102). The micrococcus catarrhalis and the meningococcus, both Gram-negative, are sometimes found in the conjunctival sac. They may be distinguished from the gonococcus by the ease with which cultures are obtained and by agglutination tests. The micrococcus catarrhalis is rarely found in acute conjunctivitis, but more often in chronic and post-operative forms.

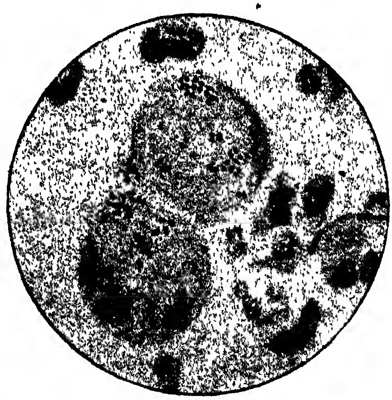


Fig. 102.—Gonococci (× 1000).

The disease is due to direct infection from the genitals. Males suffer most, the right eye before the left in a right-handed person. There is more swelling of the lids and conjunctiva than in children, copious purulent discharge, more tendency to involvement of the cornea, and marked constitutional disturbance—rise of temperature, and so on, but especially very marked depression of spirits. The greater danger to the cornea is due to the chemosis, which produces

blood and lymph stasis and facilitates the retention of secretion.

The incubation period is a few hours to three days. The upper lid becomes enormously swollen and tense, overhanging the lower, and edged with pus. Eversion, which is difficult, shows that the palpebral conjunctiva is deep red and velvety: rarely there is a membrane. Occasionally the discharge is sanious rather than purulent, especially in streptococcic cases. There is great pain; the preauricular gland is enlarged and tender, and may suppurate.

After two or three weeks the purulent discharge diminishes, but subacute conjunctivitis with much papillary thickening of the conjunctiva persists for several weeks longer. The gonococcus is still present—a point of great importance, both as regards contagion and treatment. No immunity is conferred by the attack.

The most important point in diagnosis is the coincidence of urethritis. The most important point in prognosis is the condition of the other eye, which, however, in any case usually suffers less severely.

Corneal complications are the rule, and constitute the causes of blindness. There may be diffuse haziness of the whole cornea, with grey or yellow spots near the centre. Ulcers may occur at any part, and are due to necrosis of the epithelium through direct invasion by the organisms. Marginal ulceration, which may extend completely round the cornea, is due to retention of pus in the angle formed by the chemotic conjunctiva. When ulceration has commenced it progresses rapidly and deeply, since the tissues are bereft of their first line of defence—the epithelium. Perforation is therefore common, with all its attendant dangers (*vide* p. 202). Ulceration commencing late in the history is not so dangerous.

FIG. 103.—Buller's shield.

The greatest care should be taken to prevent injury to the cornea during the manipulation necessary for diagnosis and treatment. Abrasions are easily produced by the finger nails, and even by the rough use of wool swabs. Such abrasions rapidly become dangerous ulcers.

Iritis and iridocyclitis, with attendant complications, may arise independently of perforation of the cornea, and lead to serious diminution of vision.

Gonorrhœal arthritis is not uncommon, and endocarditis and septicæmia may arise as complications.

Treatment must be directed first to protection of the other eye. Several drops of a solution of penicillin (1,000 to 2,500 units per c.c.) are instilled, and it is at once sealed up with a Buller's shield, which consists of a watch-glass stuck in a frame of adhesive plaster, or better, rubber (Fig. 103). The rubber is hermetically sealed down to the face and nose except at the lower outer angle, where a small piece of tubing is inserted under the edge. If this means of ventilation is not adopted the glass becomes hazy and the eye cannot be properly observed; moreover, the irritation to the eye is greater. Special attention should be directed to fixing the shield near the nose, *i.e.*, on the side of the source of infection. Patients should be told to sleep lying on the same side as the affected eye.

If the second eye should show signs of infection it must be treated, but every utensil or dressing which is applied to the eye must belong to a totally different set from that used for the worse eye, otherwise it may be inoculated rather than treated. The less affected eye must always be dressed first.

If pus from a gonorrhœal conjunctivitis spurts into the surgeon's eye, the conjunctival sac should be freely irrigated with sublimate lotion and penicillin solution instilled every few minutes for half-an-hour and then some half-dozen times at half-hourly intervals. The eye should be carefully watched, but no further drastic treatment applied unless conjunctivitis supervenes. The accident is due to carelessness, for every attendant on a gonorrhœal case should wear protective goggles.

If the disease is fully established and there is any purulent discharge, the eye must be irrigated with warm saline and intensive penicillin therapy started, using drops in a concentration of 2,500 units per c.c. every minute for half-an-hour. Repeated irrigations are unnecessary since in the first place penicillin remains effective in the presence of pus and in the second the discharge rapidly disappears. Any pus that does accumulate is wiped away with moist pledgets of cotton-wool. Penicillin drops are continued at five-minute intervals for a

further half-hour, and the treatment consolidated by half-hourly and then hourly instillations for two days or so. Astringent lotions are then employed (*vide* p. 173).

An alternative but less efficacious, although a useful supplementary method of treatment, is the systemic administration of a full course of sulphonamides: sulphapyridine or sulphadiazine is probably the best (*vide* p. 695).

In default of penicillin treatment most reliance must be placed on applications of silver nitrate, but they must be used with discretion. When not contra-indicated the conjunctiva of the everted lids should be well painted with silver nitrate, gr. x. to ℥ i., not oftener than once a day. Special care must be taken to avoid injury (*vide* p. 89). This treatment is contra-indicated in the very early stage before free discharge has set in, and also in later stages if there is much brawny swelling with comparatively little discharge. Under these conditions the stasis is so great that reaction is inefficient; the caustic, in fact, induces the necrosis which it should be our endeavour to avoid. In such cases hot applications and leeches should be relied upon. The latter are applied over the temporal region near the outer canthus. If the lids are very tight the outer canthus should be split (canthoplasty). The ends of strong blunt-pointed scissors are inserted between the lids into the angle under the outer canthus, which is then divided by a single snip in a horizontal direction. This has the good effect of bleeding the patient slightly, and also of giving free exit to discharge.

The eye is irrigated every two hours during the day and every four hours during the night with warm saline (0·85 per cent.), boric, or weak sublimate lotion (1 in 8,000), mercurochrome (1 per cent.), or acriflavine (1 in 1,500). Iced wet dressings are applied in the intervals, but are to be bandaged on quite loosely, so as to avoid retention of discharge. The patient is kept in bed, and if intelligent, can bathe his lids in iced lotion in the intervals. Iced applications afford much relief to the patient.

It is of great importance to attend to the general health. The bowels must be kept freely open. The strength must be reinforced by every available means—good foods, tonics, alcohol if necessary. An occasional sleeping draught and the use of sedatives must be ordered according to general prin-

ciples. Every effort must be made to combat the depression from which these patients suffer.

In the final stage of the disease silver nitrate should be used at increasing intervals for a week or fortnight after the purulent discharge has ceased. Astringent lotions are then employed (*vide* p. 173).

Atropine should be used in all cases where the cornea is involved, since this is always accompanied by some iritis; but the intraocular tension should be watched.

Corneal complications require very active treatment (*vide* pp. 204, *seq.*).

Metastatic Gonorrhœal Conjunctivitis sometimes occurs in adults, associated with gonorrhœal arthritis. It is a mild simple conjunctivitis, generally bilateral, and occasionally accompanied by iritis. It is probably due to endogenous infection from gonococci in the blood. It usually responds readily to local treatment, but is apt to recur if the arthritis relapses.

Ophthalmia neonatorum is a preventable disease occurring in new-born children as the result of carelessness at the time of birth; it used to be responsible for 50 per cent. of blind children and about 7—8 per cent. of all blind people (8 per cent. in U.S.A., de Schweinitz), but recently, although its incidence is still high, effective methods of treatment should materially reduce its occurrence and the seriousness of the sequelæ. It is difficult to obtain reliable statistics especially since all cases of discharge from a baby's eye within three weeks of birth have become notifiable as ophthalmia neonatorum (*vide* p. 162). It is obvious that a large proportion of these cases are not ophthalmia neonatorum in the narrower sense of the term. True ophthalmia neonatorum is due to infection by vaginal or fæcal matter, or from dirty rags used to clean the eyes. Purulent discharge is usually noticed on the third day; when it occurs later it is generally due to post-partum infection. In rare cases the disease is already present at birth.

Any discharge, even a watery secretion, from a baby's eyes during the first week should be viewed with suspicion, since tears are not secreted at this early date. In cases of infection the discharge rapidly becomes muco-purulent and then purulent. Both eyes are nearly always affected, though one is usually worse than the other. The conjunctiva becomes intensely inflamed, bright red, and swollen, and pours out thick

yellow pus. Marked chemosis is a distinguishing feature from severe muco-purulent conjunctivitis, and when the lids are separated by retractors the cornea is seen at the bottom of a crater-like pit. There is dense infiltration of the bulbar conjunctiva, and the lids are swollen and tense. Later the lids become softer and more easily everted, the conjunctiva becomes puckered and velvety, and the blood stasis gives place to intense congestion, with free discharges of pus, serum and often blood. In some cases a false membrane forms, so that the case resembles a membranous conjunctivitis.

There is great risk of corneal ulceration in ophthalmia neonatorum, especially, as is usually the case, when it is due to the gonococcus, which has the power of invading intact epithelium. The slightest haziness of the cornea should be viewed with apprehension. Often the cornea is already ulcerated, and not infrequently perforated, when the child comes under observation. Ulceration usually occurs over an oval area just below the centre of the cornea, corresponding with the position of the lid margins when the eyes are closed, and, consequently, rotated somewhat upwards. More rarely oval marginal ulcers are formed as in the gonorrhœal conjunctivitis of adults, or the ulceration may be central. The ulcers extend rapidly, both superficially and in depth, largely owing to lymph stasis due to strangulation of the nutrient vessels.

Perforation is usually signalised by a black spot or area in the ulcer, caused by protrusion of the iris. Sometimes perforation is sudden, a large part of the iris prolapses, and the lens may be extruded. In some cases there is a black hole in the cornea, filled with clear vitreous.

Metastatic stomatitis and arthritis occur rarely, as in gonorrhœal urethritis. The arthritic manifestations usually appear in the third or fourth week and affect knee, wrist, ankle, or, sometimes, elbow. The course is benign, abscesses being rare.

The baby's eyes must be examined as described in method (4), p. 81. The surgeon must wear protective goggles lest pus spurt into his eyes when the child's lids are separated. Retractors must always be used for separating the lids, since the slightest pressure on the eyeball may result in perforation (*vide* p. 89).

A bacteriological examination should be made in every case.

Pathology. Although probably 60—70 per cent. of cases are

due to the gonococcus, the more widespread prophylactic treatment of maternal gonorrhœa in Britain in recent years has lowered this proportion considerably. Staphylococcal infection is common, streptococcal, pneumococcal, coliform, diphtheroid or mixed infection rarer; but a large proportion of cases shows no organisms in smears or cultures and demonstrates intracellular inclusion bodies in conjunctival scrapings. It is probable that some 10 to 30 per cent. of cases are "inclusion blenorrhœa," a venereal virus infection derived from the cervix or vagina of the mother.

Sequelæ of Ophthalmia Neonatorum. In inadequately treated cases serious sequelæ may occur. If the corneal ulceration heals without perforation there is always much scarring of the cornea, but the nebula clears more in babies than in older people. Perforation may be followed by anterior synechiæ (*q.v.*), adherent leucoma (*q.v.*), partial or total anterior staphyloma (*q.v.*), anterior capsular cataract (*q.v.*), panophthalmitis, &c. Ophthalmia neonatorum is the commonest cause of anterior staphyloma.

When vision is not completely destroyed, but is very seriously impaired by the corneal opacities resulting from ophthalmia neonatorum, the development of macular fixation, which takes place during the first six weeks of life, is interfered with, resulting in the development of nystagmus (*q.v.*), which persists throughout life. Nystagmus may not become manifest until some considerable period after the ophthalmia.

Treatment. The disease is preventable; *prophylactic* treatment is therefore of prime importance. Any suspicious vaginal discharge during the antenatal period should be treated, and the most meticulous obstetric asepsis maintained at birth. The new-born baby's closed lids should be thoroughly cleansed and dried. The lids are then separated, and a drop of silver nitrate solution, 1 per cent., instilled into each eye (Credé's method). The eyes must be carefully watched during the first week.

If the disease is established the treatment should be on the lines already indicated for gonorrhœal ophthalmia; fortunately practically all types of infection in ophthalmia neonatorum, including the virus infection, are amenable to penicillin. The eyes are washed out with saline and if the cornea is involved atropine is instilled. A drop of penicillin (2,500 units per c.c.) is then instilled every minute until the discharge ceases. Any pus is meantime wiped away with cotton-wool, but pus does not tend to re-form while this treatment is in progress. After

about half-an-hour the clinical picture has altered and although there is some swelling of the lids and conjunctiva, the eye is dry. Thereafter penicillin drops are continued at 5 minute intervals for a further half-hour and subsequently at half-hourly intervals for 6 hours, and finally at hourly intervals for a further 12 hours. If it seems necessary, treatment may be continued at hourly intervals for a further 24 hours, by which time a clinical cure is usually obtained.

Before the advent of penicillin, general sulphonamide therapy was the treatment of choice: it is most efficient and should always be adopted in those rare instances in which the organism is insensitive to penicillin. Sulphathiazol and sulphadiazine are well tolerated by infants and the routine of general treatment is indicated on p. 695. It is to be noted that sulphacetamide used locally is ineffective, These drugs are a great advance over the classical methods of treatment by repeated irrigation and painting with silver nitrate. While these latter methods took weeks to check the infection, the sulphonamides take as many days: penicillin, on the other hand, *when used intensively* brings about a cure in as many hours.

The Prevention of Ophthalmia Neonatorum and Blindness due to this Cause. Though ophthalmia neonatorum is a preventable disease, and much attention has been directed to its prevention, it still remains the cause of a considerable amount of blindness, a circumstance all the more regrettable inasmuch as the disease is eminently treatable. It was made notifiable throughout England and Wales in 1914, being defined as a "purulent discharge from the eyes of an infant commencing within twenty-one days of birth." Under the rules of the Central Midwives Board every midwife is required, under severe penalties, to call to her assistance a medical man in any case of "inflammation of or discharge from the eyes (of the child), however slight," and to send notice to the supervising authority that medical help has been sought. The administrative measures ensure that midwives' cases come promptly under the supervision of medical officers who are accustomed to deal with this class of case. There is evidence that some medical practitioners fail to recognise early and notify promptly cases of ophthalmia neonatorum, with the result that efficient treatment is not begun early enough to prevent serious impairment of vision or even blindness.

Since most of the worst cases of ophthalmia neonatorum

are gonorrhœal, the combating of this disease should diminish the incidence of blindness. As already mentioned, ante-natal measures should be adopted, whenever possible, to cure disease causing abnormal vaginal discharge in the mother. Next, the greatest care should be exercised to avoid vaginal discharge reaching the eyes of the baby at birth. "As soon as the child's head is born, and if possible before the eyes are opened, its eyelids must be carefully cleansed. They should be thoroughly wiped with clean material such as cotton-wool, lint, or rag, using separate pieces for each eye. . . . When the baby is bathed the discharges with which its body is covered during labour are washed off into the bath-water. If its face is washed in this water matter may get into the eyes." (*Instructions to Midwives, by Central Midwives Board.*) If there is any suspicion of discharge, penicillin drops should be given prophylactically.

Before the advent of penicillin Credé's method of prophylaxis whereby silver nitrate drops were instilled into the baby's eyes was most successful in the hands of medical men, and is to be recommended, especially in cases where abnormal vaginal discharge is known to exist. It is not, however, to be recommended for universal use by midwives for the following reasons: (1) The midwife may be apt to think that, having dropped a little solution into the eye, she has done all that is necessary, and consequently neglect the scrupulous cleansing which is even more important; (2) the use of drugs will induce an inflammatory reaction which, on the one hand, she may mistake for the onset of the disease and notify accordingly; or (3) on the other hand, she may regard as "only a little reactionary discharge" what is really a manifestation of the disease itself; (4) the wrong solution may be used: there are several cases on record of strong nitric acid, probably supplied for urine testing, having been dropped into the baby's eyes in mistake for silver nitrate. Owing to the proved efficacy of Credé's method and improved administrative arrangements these arguments have lost some of their force.

When ophthalmia neonatorum has actually developed, a medical practitioner who has not had very thorough training and experience in the treatment of the disease should at once instil penicillin drops prophylactically and obtain the assistance of an ophthalmic surgeon. It is to be remembered that any discharge from a baby's eyes during the first week of life is pathological, and no risks should be taken. Given satisfactory home conditions, competent medical

supervision, and skilled nursing, home treatment gives the best results. In many cases these requirements cannot be fulfilled. In these cases probably the ideal method is to transfer both mother and child to a hospital where special provision is made for such cases.

Membranous Conjunctivitis (*Syn.—Diphtheritic Conjunctivitis*). As in inflammation of the throat the surface may become covered by a fibrinous membrane, so the same may occur in the conjunctiva; and just as the milder clinical varieties in the former were distinguished as croupous from the severer or diphtheritic, so also with conjunctivitis. It has been placed beyond dispute, however, that mild cases may be diphtheritic, and severe non-diphtheritic; hence it is best to speak simply of membranous conjunctivitis until a bacteriological examination has placed the matter beyond dispute. A variety of organisms other than the diphtheria bacillus, *e.g.*, pneumococcus, streptococcus, can produce a membrane, especially in weakly children, *e.g.*, after measles and scarlet fever, and in association with impetigo; these cases are sometimes called pseudo-membranous. They cannot be distinguished clinically with certainty.

Membranous conjunctivitis occurs chiefly in children, and shows all degrees of severity; it may be as virulent as the worst cases of gonorrhœal ophthalmia. It is rare in England, but it is of the utmost importance that it should be recognised when seen, not only on account of the grave danger to the eye, but also from the risks of contagion.

In mild cases there is some swelling of the lids and a mucopurulent or sanious discharge. On everting the lids the palpebral conjunctiva is seen to be covered with a white membrane, which peels off readily without much bleeding.

In severe cases the lids are more brawny: the conjunctiva is permeated with semi-solid exudates, which impair the mobility, compress the vessels, prevent the formation of a free discharge, and tend to necrosis both of the conjunctiva and cornea. Here the membrane separates much less readily, the underlying surface bleeding unless it is too infiltrated and solid. The membrane may be patchy or cover the whole palpebral conjunctiva, often beginning at the edge of the lid. It is seldom found on the ocular conjunctiva. The preauricular gland may be enlarged and may suppurate. The temperature is raised, unless the patient is in a moribund condition. Albumin is frequently present in the urine.

For six to ten days there is great peril to the cornea. Then

the sloughs begin to separate and the discharge becomes more profuse. In a few days the conjunctiva assumes a red and succulent appearance. There is danger now of adhesions forming between the palpebral and bulbar parts of the conjunctiva (symblepharon).

FIG. 104.—Diphtheria bacilli (×1000).

Post-diphtheritic paralysis, even of accommodation, is rare.

Cases of less severe but more chronic membranous conjunctivitis are occasionally met with. In them the membrane is cast off, but occurs again and again. The pathology of these cases is not understood; it has been seen as a complication of erythema multiforme.

Pathology. Competent bacteriologists have shown that there is little or no relationship between the severity of the local condition and the presence or absence of the Klebs-Löffler bacillus (Fig. 104). Only series of cases in which positive results have followed inoculation into animals are absolutely trustworthy, owing to the difficulty of distinguishing the diphtheria from the xerosis bacillus, with which it is morphologically identical. The pseudo-diphtheria bacillus forms little or no acid in culture and is not virulent for guinea-pigs, and the different varieties may be distinguished by agglutination tests. Neisser's stain (acetic methylene blue and Bismarck brown) demonstrates blue granules at the poles of true diphtheria bacilli in cultures of nine to twenty-four hours. Inoculation tests only are absolutely reliable.

Other cases may be due to the action of heat, caustics, severe atropine irritation, herpes iris, and other non-bacterial causes. Other bacteria which occasionally form membranes are pneumococcus, streptococcus, Koch-Weeks' bacillus, gonococcus, staphylococcus, Friedländer's pneumonia bacillus, bacterium coli, &c. Streptococcic conjunctivitis, a very virulent form, occurs chiefly in children associated with measles, scarlet fever, whooping cough, and influenza.

It is quite rare to obtain evidence of primary diphtheria

of the throat, though the disease may have been derived from a case of faucial diphtheria, and extension to the nose and throat by way of the lacrymal sac and nasal duct occurs. The genitalia should be examined for diphtheria or leucorrhœic discharge.

Treatment. Every case should be treated as diphtherial unless good negative evidence is afforded by films and serum cultures. In mild cases isolation need not be strict until the bacteriological report is obtained on the second day.

Local treatment consists of irrigations every two hours during the day and every four hours during the night with a bland lotion such as warm saline (0.85 per cent) or boric acid. One drop of atropin should be instilled at the commencement of treatment.

The most important general treatment is the administration of antitoxin as in faucial diphtheria. Since the antitoxin is innocuous it should be used at once in every doubtful case. In a less serious case doses of 2,000 units of antiserum should be injected into the abdominal wall; but in severe cases it is wiser to give immense doses of 4-6-10,000 units without delay, and a second injection may be given in 10 to 12 hours. Improvement generally follows in a question of hours after the first injection and corneal complications are thereby usually prevented. I have obtained benefit by frequently repeated local instillations of antitoxin, which is a rational procedure but seems to have been neglected. Special attention should be paid to the nutrition, and tonics are indicated.

Corneal complications must be suitably treated (*vide* p. 204). Antitoxins are specially useful, as may be shown by experiment. If diphtheria toxin is injected into the cornea of two rabbits and one is given an intravenous injection of antitoxin, the cornea of this animal will remain clear, whereas that of the other will become cloudy. Corneal ulceration, however, is usually due to secondary infection with pyogenic organisms. It may start at the middle or margin of the cornea and is not wholly due to interference with nutrition.

In *streptococcic membranous conjunctivitis* the danger of necrosis of the cornea and even of the death of the patient is considerable, so that immediate local and general treatment is necessary. If the organism is penicillin-sensitive constant instillations of penicillin drops as for purulent ophthalmia (*q.v.*) should be supplemented by a course of general penicillin therapy. If it is not sensitive a full course of treatment by one of the less toxic sulphonamides such as sulphathiazole or sulphadiazine should be instituted (*vide* p. 695).

Phlyctenular Conjunctivitis (*Syn.*—*Eczematous Conjunctivitis*). In phlyctenular conjunctivitis (Plate IV., Fig. 1) one or more small, round, grey or yellow nodules, slightly raised above the surface, are seen on the bulbar conjunctiva, generally at or near the limbus; they rarely occur on the palpebral conjunctiva. The disease is very frequently complicated with muco-purulent conjunctivitis, in which case the whole conjunctiva is intensely reddened. In pure phlyctenular conjunctivitis the congestion of the vessels is limited to the area around the phlyctens.

The disease is most frequent in children from five or six to ten or twelve years of age, but not very young children; it is rarely seen in adults. The children often have enlarged lymphatic glands in the neck, &c., or other signs of tubercle; on the other hand, every sign of tubercle may not infrequently be lacking: the children, however, are seldom robust. The first attack often follows an exanthem, especially measles. Rhinitis and adenoids are frequently present; signs of congenital syphilis may be found.

Phlyctens, as the name suggests (φλύκταινα, a bleb), at first much resemble blebs: it is doubtful, however, whether there is a true vesicular stage. They may be so small as to be seen with difficulty, but they usually measure about 1 mm. in diameter, occasionally reaching a diameter of 3 mm. or 4 mm. The larger ones are yellow, and have been described as pustules. In the later stages the epithelium over the surface is often destroyed, small ulcers being formed. When this occurs on the conjunctiva proper it is of little moment, since healing takes place rapidly without the formation of a scar. When it occurs on the cornea, as is very frequently the case, it is much more serious (*vide* p. 221).

Very frequently the skin of the lids and cheeks shows an eczematous condition, and eczema will be found not uncommon, if searched for, in other parts of the body, especially in the scalp. This fact has led the condition to be regarded by some as an ocular manifestation of eczema. The disease has indeed been regarded as an exanthem. It is probable that in most cases the eczema of the lids and face is secondary to the continual irritation of the skin induced by the overflow of tears and the rubbing of the wet surface with the hands.

Pathology throws some light on the disorder, though it by no means settles the causation. A simple phlycten shows in section a triangular area of intense infiltration, the apex of the triangle being towards the deeper layers. The sub-

epithelial adenoid layer normally contains a few mononuclear lymphocytes, but in the phlycten they are very numerous and closely packed together. The epithelium is intact, and it is doubtful if a vesicular stage has ever been observed.

If there is a considerable amount of conjunctivitis of the muco-purulent type, not only are lymphocytes present, but there are also many polymorphonuclear leucocytes, both in the sub-epithelial tissues and among the epithelial cells. In such cases the epithelium is quickly desquamated.

If a bacteriological examination is made, many of the organisms of muco-purulent conjunctivitis may be found. In the pure phlyctenular cases only staphylococci are found in abundance. Now staphylococci are not so common as might be expected in the normal conjunctival sac; it has, therefore, been concluded that the disease is due to staphylococcic infection. If staphylococci are rubbed into a healthy or excoriated conjunctiva a transitory redness occurs and rapidly passes off. Phlyctens have never been produced in this manner. The nearest approach to the artificial production of phlyctens has been by injecting cultures of tubercle bacilli, in which the organisms have been killed, into the veins of rabbits. It is doubtful if the infiltrates were real phlyctens in these experiments, but it is certain that the administration of tuberculin has not infrequently been followed by an attack of phlyctenular conjunctivitis in the human subject. Tubercle bacilli have never been found in the phlyctens.

Evidence has accumulated of recent years that phlyctenular conjunctivitis is an allergic condition, *i.e.*, an abnormal sensitivity to substances which are usually innocuous; *e.g.*, pollen (hay fever), drugs, &c. (*v.* p. 184), and bacterial proteins. To the last group belong chronic allergic conjunctivitis, phlyctenular disease, and possibly spring catarrh (*q.v.*). In phlyctenular conjunctivitis it is probable that sensitization has occurred to some endogenous toxin which is in most cases tuberculous (von Szili, Weekers), but may in other cases be derived from mild infections of long standing such as of the tonsils or adenoids. This view is supported by the frequent occurrence of eczema of the skin in phlyctenular disease.

The irritation of the eye leads the child to rub it vigorously. The lacrymation and rubbing cause an eczematous condition of the skin, in which the staphylococci normally present flourish and increase. These are rubbed into the eye, increasing the irritation, without being primarily responsible for the disease. Other organisms are also

rubbed in if they happen to be present; they find a suitable nidus in the debilitated conjunctiva, and an acute muco-purulent conjunctivitis is superimposed upon the phlyctenular disease.

Simple phlyctenular conjunctivitis is attended with few symptoms. There is some discomfort and irritation associated with reflex lacrymation. If there is no muco-purulent complication and if the cornea is not involved there is little or no photophobia.

Complications, however, are the rule, partly because the behaviour of the child conduces to them, partly because the favourite situation for the phlyctens is near the cornea. Here they are often astride the limbus. It has already been mentioned that the epithelium of the cornea is closely associated anatomically and developmentally with the conjunctiva. It is not surprising therefore that there is a great tendency for the superficial layers of the cornea to suffer when the conjunctiva is disordered, and this is seen *par excellence* in phlyctenular ophthalmia. The special corneal complications will be considered later (*vide* p. 221). In all such cases lacrymation is increased, muco-purulent discharge is often present, and photophobia is intense.

The term photophobia (φῶς, light; φόβος, fear; dread of light) is a misnomer. It is the term applied to the blepharospasm which is set up by the conjunctival, or more probably corneal, irritation, and which becomes greatly increased on the slightest attempt to separate the lids, especially if the attempt is made in bright light. This blepharospasm is not abolished in the dark; it is abolished by thorough application of cocaine, though this is difficult to effect. It must be concluded therefore that it is a reflex due to afferent impulses travelling along the fifth nerve, not along the optic nerve. It has been said that light acts as the stimulus to the fifth nerve endings in the cornea. There is little evidence to prove—though it is not disproved—that light can stimulate the fifth nerve endings; at the same time the fact that sneezing is often produced by exposure to very bright light may be adduced as a positive argument. It is far more probable that "photophobia" is due to a vicious circle of such a nature that movement of the lid over a spot denuded of epithelium, where the nerve endings are laid bare, causes reflex contraction of the orbicularis; this increases the irritation, increasing in turn the blepharospasm. This view is supported by the fact that there is little or no photophobia until muco-purulent conjunctivitis

has supervened, when denudation of epithelium occurs, exposing the nerve endings, which are further irritated by toxins.

Photophobia is more intense when the phlyctens are near the cornea, than when at a distance. It varies rather with their number than their size, and is extreme if they are so numerous as to form a ring round the cornea.

Temporary blindness has been observed occasionally in children after long-continued blepharospasm. It passes off in two or three weeks and is probably functional, induced primarily by the desire not to see and facilitated by the effect of prolonged pressure upon the globe by the tightly closed lids.

Severe blepharospasm makes the greatest care in the first examination imperative. The condition of the cornea is in all cases and at all costs to be placed beyond doubt. It facilitates investigation if the lids are gently separated and a drop of 2 per cent. cocaine instilled. The child is left for 5 to 10 minutes, and the eyes are then examined with all the precautions previously described (p. 88).

Phlyctenular conjunctivitis shows a very marked tendency to recur at intervals during the age period which is specially concerned. These recurrences usually take place when some intercurrent malady or defective condition in the patient's surroundings leads to lowering of vitality.

Treatment. Simple phlyctenular conjunctivitis is usually readily amenable to treatment, which must be local and general.

Local treatment consists in bathing the eyes frequently with hot boric or sublimate lotion: yellow oxide of mercury ointment, gr. iv.—viii. to ℥ i., is used, a piece the size of a hemp seed being placed within the lids three times a day; the eye is gently massaged by a finger placed upon the upper lid, moving the lid upon the globe.

Ointments are best applied on a glass rod. The child is placed upon its back on a couch and an assistant holds the arms against the body, keeping the legs still by pressure with the elbows. The surgeon separates the lids with two fingers of one hand and places the end of the glass rod carrying the ointment between the separated lids. Keeping the rod in position, the lids are allowed to close upon its end, and it is then withdrawn by carrying it outwards towards the temple. The other end of the rod and the surgeon's other hand are used for the other eye.

If there is any corneal complication, or evidence of its imminence, atropine, gr. iv. to ℥ i., is combined with the yellow ointment.

Very frequently the soddening of the skin with tears and the wrinkling of the skin through blepharospasm cause excoriations (rhagadæ) at the outer canthus. They much increase the blepharospasm and should always be looked for and treated. They are very troublesome unless attacked by cauterisation. They should be touched with the sharp point of the solid silver nitrate or the mitigated silver stick.

An efficient substitute for the yellow oxide which has fallen into undeserved disuse is finely powdered calomel, dusted into the eye, best from a camel's hair brush, which is not allowed to touch the eye. It often produces a remarkable improvement in intractable cases, but it must not be employed if iodides are being given internally; under these circumstances the unstable and extremely irritating mercurous iodide is formed in the conjunctival sac.

The blepharospasm is best treated, when severe, in the following manner. The child's face should be plunged in cold water and the mouth and nose kept under water until he struggles for breath; this is repeated three or four times—daily if necessary. The treatment is useless if not carried out ruthlessly, but no other method is so efficacious. It is not uncommon for a single application to render further treatment of the blepharospasm unnecessary.

The eyes are not to be bandaged unless corneal ulceration is so severe as to assume the preponderant *rôle*. A shade, covering both eyes and extending well over the temples, should be ordered. Smoked glasses may be substituted, but they should not be tightly fitting goggles, which will become soiled with the discharge and are cleaned with difficulty.

General treatment is never to be neglected, otherwise recurrence is inevitable. Fresh air is the best tonic, and the children should be kept out of doors as much as possible: the windows of living and sleeping rooms must be kept open. Sun or artificial light baths have proved very good and cold or sea-baths are useful. Good food, with a plentiful supply of fresh vegetables, is indicated. A calomel purge should initiate the general régime.

Cod liver oil and maltine are given in the cool weather and throughout the year if well tolerated. They may be alternated with preparations containing vitamins A and D. Phosphates and iodide of iron form substitutes or supplementary tonics.

Calcium in the form of calcium gluconate (ʒ i. ter in die) has been advocated.

General régime must be continued for a prolonged period in order to prevent recurrence, and, in any case, phlyctenular ophthalmia should be regarded as a sign of debility which requires attention (*vide* p. 223).

Simple Chronic Conjunctivitis occurs as a continuation of simple acute conjunctivitis, sometimes in spite of orthodox treatment, especially in the "gouty" type of patient. It is frequent when the cause of irritation is continuous—smoke, dust, heat, bad air, late hours, abuse of alcohol, and so on. A very common cause is the chronic reflex irritation induced by errors of refraction, overuse of the eye in bright electric light, &c. Permanent irritation from concretions (*vide* p. 189) in the palpebral conjunctiva, misplaced lashes, dacryocystitis, chronic rhinitis, &c., must be remembered and as far as possible eliminated. Unilateral chronic conjunctivitis should suggest the presence of a foreign body retained in the fornix, or inflammation of the lacrymal sac. It is often necessary to make a thorough and systematic investigation of the local and general conditions before the cause can be found. It is not infrequently associated with chronic intranasal trouble. The disease is too frequently regarded as trivial, but it is a source of great discomfort to the patient, who is duly grateful for permanent relief.

Burning and grittiness are complained of, especially in the evening, when the eyes often become red. Difficulty in keeping the eyes open is a common symptom. The lids may or may not be stuck together on waking.

The discharge is slight, most frequently subnormal, so that the edges of the lids feel hot and dry.

The eyes may look quite normal on first examination. When the lower lid is pulled down the posterior conjunctival vessels are seen to be congested, and the surface of the mucous membrane is sticky. The palpebral conjunctiva, upper and lower, may be congested, with velvety papilliform roughness. Occasionally it is succulent and fleshy.

Treatment consists in eliminating the cause and restoring the conjunctiva to its normal condition. Errors of refraction and chronic nasal catarrh are perhaps most likely to be forgotten; they should be sought out as a matter of routine. When heat is a prominent ætiological factor, *e.g.*, in cooks, spectrum blue glasses may be ordered, since they cut off the heat rays to a large extent. The treatment of the special

local conditions mentioned above will be discussed in their proper place. A gouty tendency should be treated by an appropriate régime.

Local treatment consists essentially in diminishing congestion and restoring the conjunctiva to its normal suppleness and secretory activity. It must be remembered that the condition is largely one of lack of tone, due to defective response to prolonged irritation. A stimulating treatment is therefore indicated, and is supplied by astringent applications, which not only act by relieving the congestion, but also promote a more healthy lymph flow and glandular secretion.

In mild cases weak astringent lotions suffice, *e.g.*, boric lotion with zinc sulphate, gr. i.—ii. to ℥ i., alum lotion, gr. iv. to ℥ i., &c. They should be used two or three times a day, not immediately before going to bed. Adrenaline has a transient effect in diminishing redness and itching. Boric acid ointment or sterile vaseline should be applied to the margins of the lids at bed-time. In recalcitrant cases mercury oxycyanide (1 in 5,000) may be used, followed by zinc sulphate lotion at a later stage.

In severer cases a preliminary painting with silver nitrate solution is indicated, repeated once or twice a week if necessary, or the milder protargol (5—10 per cent.) may be used. Silver preparations should not be ordered for application at home, since prolonged use may lead to staining of the conjunctiva (argyrosis).

Atropine, which is always resorted to by the inexperienced in intractable diseases of the eye, does much more harm than good. It causes great inconvenience from paralysis of accommodation, and has little effect upon the conjunctiva, such as it has being deleterious. Apart from this, it is extremely dangerous in elderly patients, who are specially liable to chronic conjunctivitis. In them, more than in others, there is grave danger that atropine may induce an acute attack of glaucoma, a disaster which it is impossible to overrate.

In the more severe cases of chronic conjunctivitis there is often an abnormal amount of secretion from the Meibomian glands. This should be squeezed out of the glands by pressure on the lid with the thumb against a spatula laid upon the conjunctival surface.

Angular Conjunctivitis (*Syn.—Diplobacillary Conjunctivitis*) is one of the few forms in which a specific organism causes a typical clinical picture. In it the reddening of the conjunctiva is limited almost exclusively to the inter-marginal

strip, especially at the inner and outer canthi, and to the bulbar conjunctiva in the same neighbourhood. Besides the conjunctivitis there is also excoriation of the skin at the inner and outer angles, which may be very slight, a mere scurfiness, but is nearly always present. After a few cases have been seen the typical picture is very easy to recognise, but the condition is not always typical. There is discomfort, with slight muco-purulent discharge; blinking is often complained of. Not infrequently there is nasal catarrh, and diplobacilli are found in the nasal secretion. If untreated the condition becomes chronic and may give rise to definite blepharitis. Clear, shallow corneal ulcers may occur, but are rare. They are usually marginal, but may be central and associated with hypopyon (*vide* p. 214). A single attack does not confer immunity, and relapses are not uncommon.

Fig. 105.—Diplobacilli (× 1000).

Pathology. The disease is due to the Morax-Axenfeld diplobacillus (Fig. 105). The bacilli consist of pairs of large, thick rods, placed end to end. They stain well with basic stains, are decolourised by Gram, and are easily recognised in films. They produce a proteolytic ferment which acts by macerating the epithelium. There is an incubation period of four days. The diplobacilli are strongly resistant to drying. They have been found in the nasal tract of healthy persons, and are often present in the nasal discharge in cases of angular conjunctivitis.

Treatment. Diplobacillary conjunctivitis responds readily to zinc salts. These may be applied in the form of boric lotion with zinc sulphate, gr. ii. to ℥ i., or as drops, preferably the former. The diplobacillus will grow in cultures containing zinc salts, and these act by inhibiting the proteolytic ferment. Boric, zinc oxide, or ichthyol (2—5 per cent.) ointment is applied to the lids at night.

Follicular Conjunctivitis occurs frequently in children and young adults, both eyes being affected. It is characterised

by the formation of small round or oval translucent bodies, 1 mm. or 2 mm. in diameter, in the lower fornix (Plate IV., Fig. 2); they are less commonly seen in the upper fornix, especially near the outer and inner canthi, never on the plica semilunaris or bulbar conjunctiva. They are raised above the surface, are often arranged in parallel rows, and consist of localised aggregations of lymphocytes—follicles, sometimes wrongly termed granulations—in the sub-epithelial adenoid layer.

Microscopically follicles are indistinguishable from the solitary lymph patches in the intestine, and often also from the follicles of trachoma (*q.v.*). They do not occur in the normal conjunctiva in man. The conjunctiva is not reddened or swollen. They persist for an indefinite time, causing few symptoms, and disappear without leaving any trace, such as scarring (cf. Trachoma).

Follicular conjunctivitis is usually due to overcrowding and living in badly ventilated rooms, especially schoolrooms, but also occurs among better class school children. Isolated follicles may occur in the outer part of the lower fornix in any chronic conjunctivitis of long standing. They may be due to over-use of atropine or eserine (*vide* p. 184). The children are seldom robust, and adenoid vegetations in the throat, which are of a similar nature, are often present. The disease is probably not contagious, and never develops into trachoma, as has been held by some observers.

The symptoms are slight, consisting chiefly of slight irritation of the eyes, worse in bright lights and after near work.

Treatment. Follicular conjunctivitis seldom requires local treatment. A weak astringent lotion may be ordered, and yellow oxide of mercury introduced within the lids once or twice a day. If the follicles are very large painting with silver nitrate solution will do good; single follicles may be touched with the alum pencil. Atropine, if in use, should be stopped or replaced by an equivalent mydriatic (*vide* p. 185).

Special attention should be directed to the refraction, and any errors corrected.

The nose and throat should be investigated and the general health and surroundings put upon a sound basis.

Trachoma (*Syn.—Granular Conjunctivitis*), once known as Egyptian ophthalmia, was spread far and wide in Europe by the French armies during the Napoleonic wars. It is a serious form of folliculosis, which is responsible for the blinding of enormous numbers of people in places where it is endemic. Both eyes are almost always affected. It may be stated at

once that in England it is a rare disease except where large numbers of Irish or aliens are herded together.

In making a diagnosis the relative frequency of various forms of disease should always be borne in mind. It is a truism, but it is often disregarded, that an unusual type of case is less likely to be a rare disease than an unfamiliar manifestation of a common one. A better class child with follicles in the conjunctiva is most unlikely to be a case of trachoma, whereas, if the child goes to school in the East End of London, where there are large numbers of trachomatous aliens, the distinction of follicular conjunctivitis from trachoma becomes a matter of great difficulty.

The changes met with in the conjunctiva in trachoma are of two types, which are often present simultaneously. The *papillary type* is not specially characteristic; it is usually a more definite form of the papillary enlargement and congestion which is met with in other severe forms of conjunctivitis. The conjunctiva covering the upper tarsus is most affected, and appears red and velvety. This condition may pass into one with more uniform jelly-like thickening. Only in the comparatively infrequent cases in which no follicles can be seen will the true disease pass wholly unsuspected.

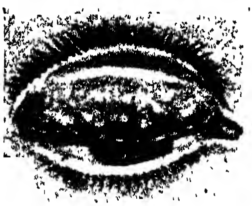


FIG. 106.—Trachoma. (After Nettleship), showing trachoma follicles and scar in typical position, parallel with the edge of the lid.

The *follicular type* (Fig. 106) manifests itself in the presence of follicles in the conjunctiva. When small they cannot be distinguished from the follicles of follicular conjunctivitis, and microscopical examination shows that they are fundamentally identical. They often, however, assume a size and appearance which is seldom or never seen in follicular conjunctivitis, but they differ most in having a characteristic distribution. The large follicles may be 5 mm. or 6 mm. in diameter. They are translucent and look like grains of boiled sago ("sago grains").

The follicles usually commence in the lower fornix, but in most cases they quickly appear in the upper also. Unlike what obtains in follicular conjunctivitis, they are not limited to the fornices. They often form a row along the upper margin of the upper tarsus, whence they invade the palpebral conjunctiva, appearing upon the tarsal surface, though they

are less numerous in this situation. They are common about the caruncle, and may be seen on the plica semilunaris. Follicles do not occur on the upper tarsal conjunctiva in follicular conjunctivitis except at the inner and outer angles. They are very rare on the bulbar conjunctiva, but when seen here they are pathognomonic of trachoma.

The disease is very chronic and leads to much irritation, photophobia, lacrymation, &c., with some muco-purulent discharge. In certain districts abroad trachoma is endemic, and a very acute form is observed. It is very doubtful if true acute trachoma is ever seen in England: what usually passes for it is of quite different origin. It is due to the lowering of resistance of the trachomatous conjunctiva whereby it becomes specially liable to intercurrent attacks of other forms of acute conjunctivitis. This is facilitated by the irritation, which leads to rubbing of the eyes, so that contamination is readily brought about. The so-called acute trachoma met with in England is therefore chronic trachoma upon which an acute muco-purulent or purulent conjunctivitis has been engrafted. Even in Egypt the acute symptoms are most often caused by the Morax-Axenfeld diplobacillus, the Koch-Weeks' bacillus, or the gonococcus (MacCallan).

Trachoma is an extremely contagious disease. Pathological anatomy reveals nothing characteristic: there is lymphocytic infiltration of the whole of the adenoid layer of the parts of the conjunctiva affected. Special aggregations of lymphocytes, without a definite capsule, form follicles which are generally indistinguishable from those of follicular conjunctivitis. In late stages and large follicles the stroma and cells tend to become hyaline and gelatinous; sometimes the surface becomes broken and the contents are extruded into the conjunctival sac. In other cases a fibrous capsule forms around the follicles, which thus become isolated: more and more fibrous tissue is laid down, giving rise to cicatricial bands such as are never formed in follicular conjunctivitis, and are very characteristic.

Noguchi isolated a *bacillus granulosus* from the trachoma of Red Indians, which when inoculated into monkeys produces follicles in the conjunctiva very similar to the follicles of human trachoma: it is probably not the cause of the disease. The "trachoma bodies" described some years ago are not pathognomonic, since such cell inclusions are very common in virus diseases of plants and animals. They suggest strongly that the disease is due to a virus, or Rickettsia, especially as

they are transmissible to lice (Cuénod and Nataf). Inclusion bodies in scrapings from the conjunctiva are therefore of some diagnostic importance.

Trachoma is endemic in many parts of the world, *e.g.*, Russia, Poland, East Prussia, parts of Austria-Hungary, Egypt, Syria, Persia, China and Japan. It shows a predilection for certain races, *e.g.*, Irish, Jews, but it is not a racial disease ; the predilection depends upon the mode of life of the individuals. Extended observation militates against the view that any race is exempt, though it is uncommon among negroes.

The disease flourishes among people who are crowded together in unhealthy rooms—in armies, navies, asylums, workhouses, schools, &c.—wherever the lower classes are herded together. Children and debilitated adults are most susceptible, but the robust are not exempt. It is commoner in low-lying, damp districts.

The disease is spread by transference of conjunctival secretion by means of fingers, towels, &c. The presence of much discharge, whether of true trachomatous origin or due to intercurrent conjunctivitis, increases the liability to contagion. On the other hand, scrupulous cleanliness suffices to prevent the extension of the disease to healthy subjects.

FIG. 107.—Trachomatous pannus. (After Nettleship.)

Trachoma in the early stages may be easily mistaken for a simple chronic conjunctivitis. This error will be avoided if it be made an invariable rule in all cases of conjunctivitis to evert the upper lids and examine the upper fornices (*vide* p. 80).

Complications. While trachoma rarely affects the bulbar conjunctiva, it not infrequently attacks the cornea.

Trachomatous pannus is a lymphoid infiltration, with vascularisation, of the margin of the cornea, usually limited to the upper half (Fig. 107), but tending to spread towards the centre and to involve the whole cornea. The upper part of the margin of the cornea becomes cloudy, and minute superficial vessels, springing from the corneal loops, grow inwards towards the centre. The haziness and vascularisation increase until the upper half of the cornea is affected. The vessels are all superficial (*vide* p. 90), and microscopic examination has

shown that they lie at first between Bowman's membrane and the epithelium. They carry in with them a small amount of granulation tissue. In later stages Bowman's membrane disappears and the superficial layers of the substantia propria become involved.

In progressive pannus the vessels are mostly parallel to each other and directed vertically downwards, anastomosing little. They extend to a level which forms a horizontal line, and beyond this line there is a narrow strip of infiltration and haze. In regressive pannus, on the other hand, the vessels extend a short distance beyond the area which is infiltrated and hazy: this difference is useful in estimating the results of treatment.

In more severe cases the vascularisation is not limited to the upper part, but superficial vessels grow in from all sides and the whole cornea becomes vascularised and opaque.

Pannus is not due to the rough upper lid rubbing upon the cornea. This is doubtless a predisposing factor, but in many conditions in which the lids are rough from some other cause pannus does not occur.

It may resolve completely, leaving the cornea quite clear, but only in cases treated early, when the vessels have not yet destroyed Bowman's membrane. In other cases a permanent opacity results. Occasionally the corneal substance becomes weakened so that the cornea bulges under normal intraocular pressure and ectasia follows (keratectasia).

Corneal Ulcers are commonest at the advancing edge of the pannus. They are shallow, little infiltrated, and very irritable, causing much lacrymation and photophobia. Indolent central ulcers may form, or there may be ulcers in any part of the cornea, but especially over the pannous area.

Sequelæ. Apart from the results of pannus and corneal ulceration the most malign effects of trachoma are caused by distortions of the lids. A peculiar drooping of the upper lids is very characteristic (*trachomatous ptosis*). It gives a sleepy appearance to the patient. There is always some scarring (Fig. 106), and when this is extensive the shape and position of the lids, especially the upper, are altered. Pressure on the everted upper lid will cause the appearance of white bloodless areas which may be mistaken for trachomatous scarring. They are easily distinguished by relieving the pressure. Through the great swelling of the conjunctiva the lids may be turned outwards (ectropion). In the late stages the follicles invade the tarsus, causing softening and absorption of the dense fibrous tissue; through the later contraction of the new-

formed scar tissue the lids may be turned inwards (entropion), causing the lashes to rub against the cornea (trichiasis), &c. (*See* Chap. XXXI.)

Treatment. The sulphonamide drugs have shown themselves to be of great value in the treatment of trachoma. As a rule they are not effective against virus infections, but it is now apparent that they are effective against the class of large-sized viruses to which the infective agent of trachoma appears to belong. Treatment should be both local and general. A full course of sulphonamide therapy should be undertaken (*vide* p. 695) and simultaneously the lids should be painted once or twice a day with sodium sulphacetamide (30 per cent. solution) and sodium sulphacetamide ointment (6 per cent.) instilled in the intervals. This combined with mechanical expression of the follicles in florid cases usually brings about cure more effectively and more rapidly than the classical and more drastic method of therapy by copper sulphate or silver nitrate. In some cases blepharospasm has been relieved within twenty-four hours, and inclusion bodies have disappeared in three days: the bulbar conjunctiva becomes white in a few days, pannus is reduced, and corneal ulcers may be healed in a week. Relatively avascular lymph follicles, however, persist and are absorbed slowly.

In most cases of trachoma the treatment advised will suffice to bring about that condition of amelioration which is usually described as cure. Relapses are common, occurring sooner or later, according to the length and assiduity of treatment.

Useful alternative applications which may be used in addition to local sulphonamides are painting with a saturated solution of quinine bisulphate (2 per cent. or mercury perchloride used as a paint in a 1 per cent. or 2 per cent. solution in glycerine and applied to the fornix and everted lids. This acts as a protoplasmic poison, is followed by an intense reaction and the treatment is almost unbearably painful, the conjunctiva and lids becoming enormously swollen. The pain lasts for several hours, gradually diminishing in intensity. Ice, or lint wrung out in iced water, should be applied to the lids immediately after the application. There is no doubt that much benefit is derived from the treatment.

The classical treatment consisted of scouring the conjunctiva with a smooth crystal of copper sulphate fixed in a wooden holder and

pointed at the end. The lids are everted, and the point of the copper stick is pushed well up into the upper fornix and moved from one side to the other, the lid being lifted away from the globe by the stick during the manœuvre so as to avoid touching the cornea. The stick is then rubbed firmly over the whole of the palpebral conjunctiva. The application of the copper stick is very painful, especially during the first few applications. Pantocain may be previously instilled, but it does not prevent the intense smarting. It is important to start with the upper fornix, since this is most affected and most difficult to reach; if it is not done first blepharospasm and the struggles of the patient will make it impossible afterwards. It is useless to apply copper too gently; it must be firmly rubbed into the conjunctiva. Some of the sulphate dissolves in the tears, and should be mopped up with a pad of dry wool, since it is very irritating to the cornea. Pannus is no contra-indication to the use of the copper stick, but quite the reverse; copper sulphate applied to the lids is the best treatment for pannus. The pannus itself is not treated directly at all. On the other hand, corneal ulceration of any kind or degree is an absolute contra-indication.

The follicles may also be destroyed by touching them with a solid stick of carbon dioxide snow. Care must be taken to allow the tissues to thaw before the everted lid is replaced. X-rays and radium have also been used, but do not give better results than the ordinary treatment.

When the follicles are numerous and very prominent the treatment is shortened by attacking them mechanically. This may be done by various forms of scarification or expression. In performing any operation upon trachomatous patients protective goggles must be worn by the surgeon and the immediate attendants.

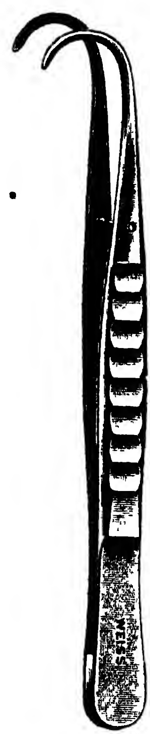
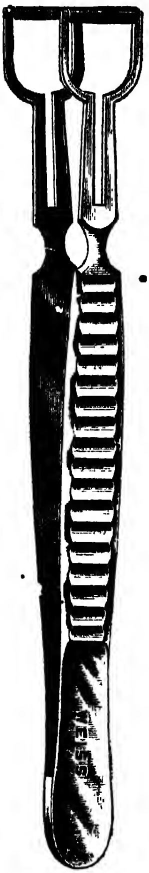


Fig. 108.—Graddy's forceps.

The conjunctiva is first thoroughly anæsthetized. Scarification may be performed by a knife, needle, sharp spoon or stiff toothbrush. The follicles may also be destroyed by the galvano-cautery or by electrolysis.

In *expression* one of the many forms of expressor is used—*e.g.*, Graddy's forceps (Fig. 108), Knapp's roller forceps (Fig. 109). The upper lid is everted, and one limb of the forceps is pushed up into the fornix, the other being laid upon the palpebral conjunctiva. The two limbs are then pressed together with moderate force and are drawn in a horizontal direction from one end of the fold to the other. The fold of the fornix should in this manner be thoroughly mangled, and all the follicles squeezed out.



FULL SIZE

FIG. 109.—Knapp's roller forceps.

If the follicles in the upper fornix are very large and closely packed it is well to commence treatment by excising the fornix. There is always a redundance of tissue here, and no evil results ensue. The upper lid is everted doubly (*vide* p. 81) so as to expose the retro-tarsal fold completely. A silk suture is then passed through the fold at each end. By dragging on the sutures the whole fold is drawn out. It is then excised with scissors. If the tarsal plate is much diseased or distorted it also may be excised.

Pannus requires no special treatment, but if it fails to respond a *peritomy* may be performed. In this operation a collar of conjunctiva, 5 mm. broad, is excised round the corneal margin. The raw surface of the sclera should be seared with the galvano-cautery, the object being to destroy the vessels and prevent their reformation. Corneal ulcers must be treated on general principles (*vide* p. 204).

Inclusion Blennorrhœa is an acute follicular conjunctivitis caused by a virus disease, as shown by the presence of inclusion bodies and by the transference of the disease by the filtrates of emulsified conjunctival scrapings through a fine Berkefeld filter. It is the cause of *swimming-bath conjunctivitis*, but is also found in inmates of schools, asylums, &c. Its occurrence in cases of ophthalmia neonatorum and the presence of inclusion bodies in urethral and vaginal discharges suggests a genital origin. It is amenable to local treatment by penicillin or systemic treatment by the sulphonamides.

Tubercle of the Conjunctiva is rare: it nearly always produces ulceration. Conjunctival ulceration should always suggest either the presence of an imbedded foreign body or a tuberculous or syphilitic lesion.

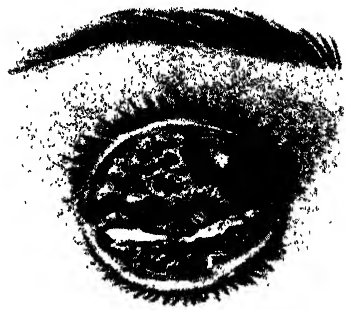


FIG. 110.—Tubercle of the conjunctiva. (After Eyre.)

Tubercle occurs in several forms: (1) small miliary ulcers usually on the palpebral conjunctiva; (2) granules on the palpebral conjunctiva resembling trachoma follicles (Fig. 110); (3) gelatinous cockscomb-like excrescences on the fornices; (4) polypoid pedunculated outgrowths; (5) a solitary nodule near the limbus, which may become infected with pyogenic organisms and ulcerate. Occasionally tubercle attacks the bulbar conjunctiva elsewhere, and the conjunctiva may be affected by extension of lupus from the face. These cases must be distinguished from those with secondary extension of tubercle from within the eyeball (*vide* pp. 267, 342). Infection is generally endogenous.

The preauricular gland is often enlarged and may suppurate. The disease is chronic, and the ulcers are indolent. The patients are usually young, and often free from clinical signs of active tuberculous disease elsewhere in the body. There is little doubt that the lesion may be the primary seat of tubercle, the bacilli being inoculated into minute abrasions, which are probably always present in the conjunctiva, caused by dust. There is little pain or irritation unless the ulceration is extensive.

It is not improbable that the second type is frequently mistaken for trachoma, and it is possible that it is cured by the treatment founded on the wrong diagnosis. I have seen cockscomb tubercle in the lower fornix associated with tuberculous disease of the lacrymal sac in a child.

Pathology. Scrapings may show tubercle bacilli. Sections show typical giant-cell systems. In doubtful cases inoculation experiments should be made. If a piece of tuberculous tissue is introduced into the anterior chamber of a rabbit's eye

a typical tuberculous iritis will ensue in two or three weeks. Intraperitoneal inoculation of guinea-pigs is more satisfactory. Dermal tests or injections of tuberculin may be tried.

Treatment. The disease should be eradicated, more especially as being often the primary focus. The affected conjunctiva should be excised, or if this is not feasible, thoroughly scraped and cauterised.

Injections of tuberculin have given encouraging results, and subconjunctival injections of 2 per cent. guaiacol cacodylate may be employed. The application of 50 per cent. solution of lactic acid has been advocated. Local ultra-violet ray therapy is certainly beneficial.

Extension to the globe is probably rare. If prolonged treatment fails and the disease spreads it may be necessary to excise the eye. Cases of lupus should be treated with calciferol.

Syphilis manifests itself rarely in the conjunctiva in the form of a primary chancre, which is less indurated than the ordinary genital chancre, or gummatous ulceration. In the former case it may be due to the removal of a foreign body with the tongue. Ulceration of the palpebral or still more of the bulbar conjunctiva is always suggestive of the condition. Scrapings should be taken and examined for spirochætes, and the Wassermann test should be applied (*vide* p. 622). A primary chancre of the palpebral conjunctiva may be wrongly diagnosed and treated as a chalazion (*vide* p. 625).

Tuleremia. Tuleremia is a recently elucidated disease with a widespread distribution in America, Europe and Asia caused by an organism (Bacterium Tulerense) derived from animals, particularly squirrels and rabbits. In the oculo-glandular form ulcers and nodules appear on the tarsal conjunctiva associated with the pre-auricular lymph glands and associated with general constitutional symptoms of fever and debility. The diagnosis is made by an agglutination test and treatment is symptomatic.

Conjunctivitis caused by Drugs (Atropine Irritation, &c.) and Irritants. Some people are particularly susceptible to atropine, and more rarely to eserine and other drugs, locally applied to the conjunctiva. The lids become swollen, tense, and red, in fact erysipelatous. Examination of the conjunctiva will often show follicles, and even rarely a membrane. The dust of teak wood is particularly irritating to the conjunctiva. Conjunctivitis is also caused in some people by contact with horses or cats, or certain flowers, especially *Primula obconica.* Most

of these and other irrritative reactions are manifestations of allergic hypersensitivity, comparable to hay fever.

Workers with chrysophanic acid suffer from conjunctival irritation, and prolonged internal administration of arsenic causes the same effect. A chip of aniline pencil in the eye causes much irritation and unsightly staining, and may lead to ulceration and necrosis. Some eyelash dyes cause severe conjunctivitis and dermatitis.

Malingerers sometimes induce conjunctivitis by the insertion of tobacco, ipecacuanha powder, &c., into the eyes. The irritation is most marked in the lower fornix, and usually the right eye is affected in right-handed people.

Treatment. In atropine irritation the drug should be avoided. If a mydriatic is imperative, some other should be substituted, *e.g.*, duboisine sulphate, 0·5 per cent.; scopolamine hydrobromide, 0·25 per cent. to 0·5 per cent., or hyoscine, 0·5 per cent., may be used, but toxic symptoms sometimes occur. Subconjunctival injection of mydricain (*vide* p. 694) may be used with impunity in these cases. Atropine irritation has been cured in some cases by instilling adrenaline (1 in 1,000) drops, moistening the skin of the lids with this solution, and subsequent application of zinc oxide ointment (Wolff). The conjunctiva soon recovers after cessation of the cause, but astringent lotions accelerate the cure.

Aniline dye staining can be removed by washing out with weak alcohol solution and instillation of weak glycerine drops (Werner).

Action of Caustics. See p. 435.

Poison gases used in warfare include lacrymatory gases, phosgene, mustard gas, arsenicals, and other secret agents.

The *lacrymatory gases* include K.S.K. (ethyliodoacetate), B.B.C. (bromobenzylcyanide) and C.A.P. (chloroacetophenone). They cause immediate irritation of the eyes, profuse lacrymation and blepharospasm. The conjunctiva is injected and swollen, but there is no involvement of the cornea. The symptoms disappear in a few hours, and the eyes respond well to lavage with bland lotions. Chlorine, phosgene, arsenical compounds and "smokes" also cause conjunctival irritation.

Mustard gas (dichlorethyl sulphide) usually produces ocular symptoms after a latent period of from 6 to 8 hours, and is effective when very dilute (1 in 10,000,000 in air). In moderately severe cases the conjunctiva is congested and swollen in the interpalpebral area. Functional blepharospasm may persist after all inflammatory signs have disappeared, and fear of blindness may delay convalescence. In more severe cases the interpalpebral zone of conjunctiva is white from coagulated exudate, and chemotic conjunctiva bulges forwards from the fornices. The lids are swollen and stuck together by discharge. The cornea is stippled ("orange-skin" cornea), with œdematous and roughened epithelium, and corneal

sensation is diminished; or the interpalpebral strip may necrose. A considerable number of cases break down with recurrent corneal ulceration several years later.

Arsenical vesicants are much more destructive in their action. They cause rapid necrosis of the conjunctival and corneal tissues with which they come into contact resulting in dense corneal opacities or even perforation of the eye.

The *treatment* of conjunctivitis caused by lacrymatory gases is irrigation with bland lotions—normal saline, boric or sodium bicarbonate (2 per cent.). The eyes should not be bandaged, but dark glasses used.

In mustard gas injuries the lids should be gently separated, the cornea inspected and the patient assured that he is not blind. The eyes are irrigated with warm sodium bicarbonate lotion and cod-liver oil drops instilled; dark glasses are worn, and vaseline ointment is applied to the lid margins at night. If the cornea is hazy, or stains with fluorescein, atropine (1 per cent.) should be instilled twice daily, with the same precautions as described for hypopyon ulcer (*vide* p. 214). Sulphonamides may be given by the mouth if there is severe secondary infection. Tarsorraphy may be necessary in some cases. It is very important to combat the neurasthenia by appropriate means. At a later stage contact glasses afford protection to the cornea and aid vision.

Arsenical vesicants are neutralised by the local application of B.A.L. ointment (British Antilewisite) which is effective if it is applied within a few minutes of the injury.

Spring Catarrh (*Syn.—Vernal Conjunctivitis*). This is a recurrent conjunctivitis occurring with the onset of hot weather, and therefore rather a summer than spring complaint. It is found in young people, nearly always boys, associated with the usual symptoms of mild conjunctivitis. Burning, itching, some photophobia, and lacrymation are the chief symptoms. Both eyes are affected. In the cooler months the condition subsides and gives no trouble, but recurs with the return of heat. The disease is met with among all classes, is sporadic, and non-contagious. It has been attributed on insufficient grounds to the action of the actinic rays of the spectrum. It is more probably an allergic condition, as indicated by the accompanying eosinophilia.

Two types of objective signs are met with: (1) the palpebral form; (2) the bulbar form: both may be combined, but this is relatively rare. The palpebral form is easily recognised if typical. On everting the upper lid the palpebral conjunctiva is seen to be hypertrophied and mapped out into polygonal raised areas, not unlike cobble stones (Fig. 111). The colour is bluish white, like milk, and this appearance is seen also over the lower palpebral conjunctiva.

The flat-topped nodules are hard, and consist chiefly of dense fibrous tissue, but the epithelium over them is thickened, giving rise to the milky hue. In vertical section they resemble circumvallate papillæ. Eosinophile leucocytes are present in them in great numbers and are found in the secretion.

The palpebral form cannot be mistaken if typical, but it may resemble trachoma. The type of patient, the milky hue, the freedom of the fornix from implication, and the characteristic recurrence in hot weather will usually prevent mistake.

The bulbar form is less characteristic. In it there is a wall of thickening at the limbus, more gelatinous in appearance, and also milky. It may be mistaken for phlyctenular conjunctivitis.

In both forms the lesions persist during the cold months, though they are less marked.

Serious complications never supervene, and the ultimate prognosis is good, though recurrences may persist for several years. Occasionally a peculiar corneal opacity, resembling arcus senilis in having a clear zone between it and the limbus, is left, and

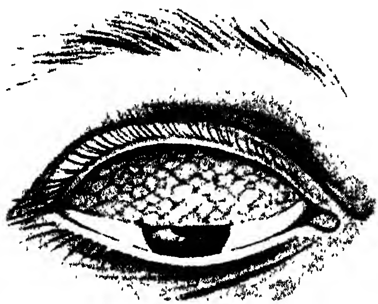


FIG. 111.—The palpebral form of spring catarrh.

some thickening and discoloration of the conjunctiva may remain.

Treatment is purely symptomatic. Well-fitting goggles with tinted glass should be worn. The irritation is best relieved by very weak acetic acid, gt. i. to ℥ vi. Adrenaline gives temporary relief. Gentle massage with the upper lid after application of yellow oxide of mercury ointment or 1—2 per cent. guaiacol ointment is beneficial. Astringents are harmful: boroglyceride has been recommended, with arsenic internally. The application in 10 milligrams of unscreened radium (β rays), held by a layer of varnish in the shallow trough of a monel metal spatula shaped for insertion under the upper lid, at monthly intervals during February, March and April, seems to be of value in preventing an attack, but does not cure the disease. Excision of the nodules, sometimes advised, seems to be useless. The general health

should be attended to, and adenoids and enlarged tonsils, if present, removed.

Ophthalmia nodosa is a nodular conjunctivitis which may be mistaken for tubercle—pseudo-tuberculous disease of the conjunctiva. It is due to the irritation of the hairs of certain caterpillars, and therefore always commences in the late summer months. Small semitranslucent, reddish, or yellowish grey nodules are formed in the conjunctiva and sometimes in the iris. On microscopical examination hairs surrounded by giant cells and lymphocytes are found.

Treatment. The nodules in the conjunctiva should be excised. Otherwise the condition is treated on general principles.

Parinaud's Conjunctivitis may be mistaken for tubercle or trachoma. Usually one eye only is affected. Granulations occur on the tarsal conjunctiva or fornices, and the preauricular and submaxillary glands are enlarged and may suppurate. The disease commences with slight constitutional disturbance and may last for months. It has been attributed to bovine tubercle and to a leptothrix (Verhoeff).

Pemphigus of the conjunctiva is a rare but very serious disease affecting both eyes. Vesicles occur, but more commonly greyish white membranous patches. Progressive cicatrisation of the conjunctiva follows, leading eventually to *essential shrinking of the conjunctiva,* with consequent opacification of the cornea from malnutrition. Vesicles may be found in the nose, mouth and pharynx, but rarely in the skin. Treatment, such as transplantation of mucous membrane, is unavailing. Contact glasses applied with parolein may give some relief and improve vision.

Electric Light Ophthalmia (Photophthalmia). Ultra-violet rays may cause extreme burning pain, lacrymation, photophobia, blepharospasm, and swelling of the palpebral conjunctiva and retrotarsal folds. There is always a latent period of four or five hours between the exposure and the onset of symptoms. The condition is generally caused by the bright flash of a short circuit, but may result from exposure to a naked arc light, as has happened, for instance, in cinema studios. It is rarely due to exposure to enclosed arc or other lights, since the glass globe absorbs the most deleterious of the ultra-violet rays. Eosinophile leucocytes are increased in the secretion.

Treatment. Cold compresses, dark glasses, and astringent lotions.

Snow Blindness. The cause and symptoms of snow blindness are the same as of electric light ophthalmia, viz., expo-

sure to ultra-violet rays, especially from 311 $\mu\mu$ to 290 $\mu\mu$, an unusually large percentage of which is reflected from snow surfaces. Smoked or orange tinted glasses should be used as a prophylactic measure, and they are most efficacious when made with Crookes's glass (*vide* p. 148). The treatment is the same as for electric light ophthalmia.

Degenerative Changes in the Conjunctiva

Concretions (*Syn.*—"*Lithiasis*"). Concretions occur as minute hard yellow spots in the palpebral conjunctiva. They are due to the accumulation of epithelial cells and inspissated mucus in depressions which are called Henle's glands. They never become calcareous, so the term is a misnomer, but they are so hard that when they project from the surface they scratch the cornea and give the sensation of a foreign body in the eye. They are common in elderly people. There is no reason to attribute them to gout, but uric acid deposits have been observed in the palpebral conjunctiva of gouty patients. Concretions should be removed with a sharp needle.

Pinguecula is a triangular patch on the conjunctiva, found usually in elderly people, especially those exposed to dust, wind, and so on. It occurs in the direction of the palpebral aperture, the apex of the triangle being away from the cornea. It affects the nasal side first, then the temporal. It is yellow in colour and looks like fat, whence the name (pinguis, fat). It is not due to fat, but to hyaline degeneration of the connective tissue and an excessive development of yellow elastic fibrous tissue. It is particularly conspicuous when the eye is inflamed, since the pinguecula remains relatively free from congestion: mistakes in diagnosis may then occur. It requires no treatment, but may be removed if the disfigurement is great.

Pterygium (πτέρυξ, a wing). This is a peculiar encroachment of the conjunctiva on the cornea (Fig. 112). It is triangular in shape, and when single is always upon the nasal side; when double the temporal one has developed later. It is derived from pinguecula (Fuchs). It must be carefully distinguished from *pseudo-pterygium*, which is due to the tip of a fold of chemotic conjunctiva becoming adherent to an ulcer within the corneal margin. It may occur at any part of the cornea. The conjunctiva then forms a bridge over the limbus, and a fine probe can always be passed beneath it, which is not possible with a true pterygium.

The apex of the pterygium is usually blunt; there is no ulcer in the cornea beyond it, as formerly described, but there are often small opacities. In the early stage the pterygium is thick and vascular; it advances over the cornea and may reach the pupillary area and interfere with vision. When it ceases to grow it becomes thin and pale, but never disappears.

The true pterygium is a single layer of conjunctiva, adherent in its whole length to the sclerotic and cornea, though only loosely, except at the apex. The area of adhesion is always smaller than its breadth, so that there are folds at the upper and lower borders.

Pterygium is not due to a fold of conjunctiva being dragged across the cornea by a progressive ulcer, as was once taught.

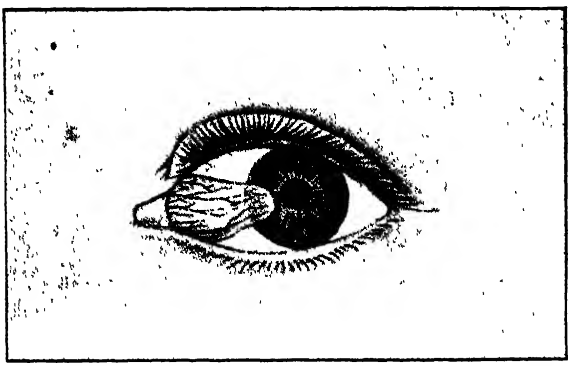


FIG. 112.—Pterygium.

It is probably due to malnutrition of the cornea, resulting from the pinguecula, and prolonged irritation. Granulation tissue grows in under the epithelium, destroying Bowman's membrane. When it ceases to progress dense fibrous tissue is formed.

Pterygium is rare in England, but is common in dry climates with sandy soils, *e.g.*, parts of Australia, South Africa, Texas, &c. It was common in soldiers during the Boer War, and it is not infrequently seen in sailors.

Treatment. Pterygium is best left alone unless it is progressing rapidly towards the pupillary area, or is very disfiguring. The latter reason is not of much weight, since it cannot be removed without leaving a scar.

The apex of the pterygium may be destroyed by diathermy. Removal is effected by seizing the neck, near

the corneal margin, with fixation forceps, raising it, and shaving or dissecting the apex from the cornea. Care must be taken not to go too deep. The pterygium is freed from the sclerotic for about half the distance towards the canthus. Two converging incisions with scissors separate the apex and greater part of the body. The conjunctiva is then freed from the sclerotic above and below so as to admit of the two edges being sutured together.

Pterygium sometimes recurs after removal. This may be only apparent, owing to vascularisation of the denuded area. If it actually re-forms and extends towards the pupillary area, the apex should be turned down under the bulbar conjunctiva and sutured in this position (McReynolds).

Symptomatic Conditions

Subconjunctival Ecchymosis, due to the rupture of small vessels, frequently occurs. It may be the result of direct injury, or, more commonly, occur spontaneously. Very minute ecchymoses, or possibly thromboses, are seen in severe conjunctivitis, especially pneumococcic. Larger ones accompany severe straining, especially in old people, *e.g.*, on lifting heavy weights, vomiting, &c. In these circumstances they indicate a weakness of the vessel walls, and should be regarded as a danger signal. Other signs of arteriosclerosis should be sought in the fundus oculi and elsewhere, and, if found, warning of the possibility of cerebral hæmorrhage given, with appropriate instructions for its avoidance. Very frequently no such signs can be discovered, and the condition, though unsightly, is trivial. Subconjunctival ecchymoses are not infrequently seen in children with whooping cough ; as a rule they need arouse no anxiety, but retinal hæmorrhages and hyphæma also occur in association with them, as well as cerebral hæmorrhage.

More serious are the large subconjunctival ecchymoses which sometimes follow blows or falls on the head. They may then be due to extravasation of blood along the floor of the orbit, the result of a fractured base. In fractures of the sphenoid the blood appears later on the temporal side than elsewhere. Hæmorrhage also results from severe or prolonged pressure on the thorax and abdomen, as in persons squeezed in a crowd.

Subconjunctival hæmorrhages may occur in scurvy, purpura, malaria, and so on.

The importance of subconjunctival hæmorrhage is always symptomatic, since the eye itself is never endangered.

Treatment. The blood becomes absorbed in from one to three weeks without treatment. Boric lotion is usually ordered as a placebo. Dionin may be used to accelerate absorption in young patients, but should not be employed in elderly subjects with arteriosclerosis.

Chemosis, or œdema of the conjunctiva, may occur in (1) acute inflammations; (2) in cases of obstruction to the lymph flow; (3) in abnormal blood conditions.

In the first group of cases the inflammation may be in the conjunctiva, *e.g.*, gonorrhœal conjunctivitis, or within the eyeball, as in panophthalmitis, hypopyon ulcer. It is also found in acute glaucoma. The inflammation may be in the accessory structures of the eye, *e.g.*, a stye, a parasitic bite on the lid, dacryocystitis, periostitis, orbital cellulitis, cerebro-spinal meningitis. The chemosis of dionin is probably due to a specific irritation of the conjunctival vessel walls.

In the second group the pressure of an orbital tumour may so interfere with the lymph and blood streams as to produce chemosis, and it is found in pulsating exophthalmos.

To the third group belong Bright's disease and the anæmias It is sometimes due to urticaria or angioneurotic œdema. Recurrent chemosis is sometimes associated with menstruation, and it has been observed in trigeminal neuralgia and migraine.

Xerosis (*Syn.—Xerophthalmia*) (ξηρός, dry) is a dry, lustreless condition of the conjunctiva which occurs in two groups of cases: (1) as a sequel of local ocular affection; (2) associated with general disease.

The first type is a cicatricial degeneration of the conjunctiva —(a) following trachoma, burns, pemphigus, diphtheria, &c., commencing in isolated spots, ultimately involving the whole conjunctiva and cornea; (b) following exposure, due to ectropion or proptosis, whereby the eye is not properly covered by the lids. As the result of the rare affection of the conjunctiva with pemphigus the cicatricial contraction of the conjunctiva may be extreme and progressive, a sort of keloid condition being induced (*essential shrinking of the conjunctiva*): the lids may then be quite adherent to the globe, the cornea being opaque like skin (*vide* p. 188).

In the other group of cases xerosis occurs in a mild form, found in children, usually boys, accompanied by night blindness (Chap. XIX.), and characterised by small triangular

white patches on the outer and inner sides of the cornea, covered by a material resembling dried foam, which is not wetted by the tears (Bitôt's spots). The cases usually occur during the summer months, and the children are not conspicuously ill-nourished. A similar mild form, also associated with night blindness (*vide* p. 417), is met with in adults in some countries, *e.g.*, India, but seldom if ever in England. A severe form is found in marasmic children, associated with keratomalacia (*vide* p. 224) and necrosis of the cornea.

Pathology. The chief changes are in the epithelium, which becomes epidermoid, *i.e.*, exactly like that of skin, with granular and horny layers. The foamy spots are due to horny epithelium, which becomes cast off into the conjunctival sac and accumulates in the lower fornix. Owing to this change the epithelium ceases to secrete mucus. It becomes dry; a certain amount of vicarious activity is set up in the Meibomian glands (Chap. XXXI.), which cover the surface with their fatty secretion. The watery tears then fail to moisten the conjunctiva. The so-called xerosis bacilli, which are pseudodiphtheria bacilli, grow profusely under these conditions, but they have no causal relationship and are of no importance.

Xerophthalmia, and the associated night-blindness, is a deficiency disease due to absence of fat-soluble vitamin A in the diet. This avitaminosis is associated with demyelination of the Vth nerve in animals, so that the condition is probably essentially neurotrophic (Mellanby).

It is to be noted that xerosis has nothing to do with any failure of function on the part of the lacrymal apparatus. The conjunctiva can be quite efficiently moistened by its own secretions alone. If the lacrymal gland is extirpated xerosis does not follow. If on the other hand the secretory activity of the membrane itself is impaired xerosis follows, in spite of normal or increased lacrymal secretion. The tears of rabbits fed on vitamin A deficient diets are deficient in lysozyme (Findlay), thus explaining the susceptibility to local bacterial invasion.

Treatment. Xerosis is a symptom, and its treatment must therefore be purely symptomatic. Local treatment consists in relieving the dryness with parolein, olive oil, milk, weak alkaline solutions, &c.: smoked glasses should be worn.

In xerophthalmia restoration of normal nutrition by administration of vitamin A and other measures is all important, though in marasmic cases the patients have often gone too far.

Argyrosis is the staining of the conjunctiva from prolonged

application of silver salts (nitrate, protargol, &c.) for the treatment of chronic conjunctivitis, and especially trachoma. The conjunctiva, particularly the lower fornix, is stained deep brown. The staining is due to the impregnation of the elastic fibres in the membrane and vessel walls with reduced metallic silver. The condition is very difficult to get rid of, but subconjunctival injection (0·3 c.c.) of 2 parts of 2 per cent. potassium ferricyanide with 1 part of 12 per cent. sodium thiosulphate appears to be most efficacious: a platinum needle must be used to avoid staining with iron.

Cysts and Tumours

The only common cysts found in the conjunctiva are due to dilatation of lymph spaces. When small these often form rows of little cysts on the bulbar conjunctiva (*lymphangiectasis*). Occasionally single, though multilocular, cysts occur (*lymphangioma*). Larger retention cysts of Krause's accessory lacrymal glands occur in the upper fornix (Chap. XXXI.). Subconjunctival cysticercus and hydatid are rare in England. Non-parasitic cysts require simple removal of the anterior walls. *Epithelial implantation cysts* occur rarely after injuries or operations, *e.g.*, tenotomy, and may burrow into the cornea.

Tumours of the conjunctiva have all a tendency to be polypoid, owing to the perpetual movements of the globe and lids.

Papillomata occur at the inner canthus and in the fornices. In the latter position they may be mistaken for the cockscomb type of tubercle, but the individual leaflets may be separated by a probe. They also occur sometimes at the limbus in old people and are then liable to become malignant. They should be removed.

Simple Granulomata, generally polypoid, often grow from tenotomy wounds or the sites of foreign bodies. They consist of exuberant granulation tissue. They are common in empty sockets after excision, and at the site of chalazions which have been insufficiently scraped (Chap. XXXI.). They should be removed by scissors.

Fibromata, also generally polypoid, occur in sockets. They may be soft or hard, and require simple removal.

Nævi or *congenital moles* are not uncommon. They are white gelatinous or pigmented nodules situated by preference at the limbus or near the plica semilunaris. They have the same structure as in the skin—groups, often alveolar, of "nævus cells" in close connection with the epithelium. They are congenital and tend to grow at puberty, rarely becoming malignant. They may be excised. Pigmentation at the limbus occurs normally

in dark races, and patches in this situation are not uncommon in people with dark complexions.

Dermoids are lenticular yellow tumours, usually astride the corneal margin, most commonly at the outer side (Fig. 113). They are often wrongly called dermoids of the cornea. Not infrequently there is a notch in the upper lid corresponding with the position of the tumour. They consist of skin in an abnormal situation, with epidermoid epithelium, hairs, sebaceous glands, &c. They are congenital and tend to grow at puberty; the hairs also grow and often cause irritation. Dermoids should be dissected off the globe if troublesome, though their removal does not produce

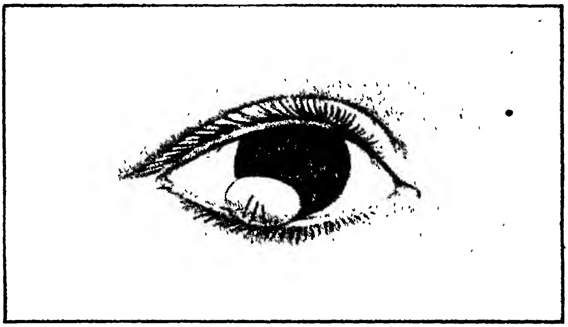


FIG. 113.—Dermoid of the conjunctiva, in a somewhat unusual situation. Note the hairs.

much improvement in appearance, as the site of attachment to the cornea remains densely opaque.

Dermo-lipomata or *fibro-fatty tumours* are congenital tumours found at the outer canthus in babies. They consist of fibrous tissue and fat, sometimes with dermoid tissue on the surface. They are not encapsuled. The main mass should be removed, but it will be found that the fat is continuous with that of the orbit: care must be taken not to injure the extrinsic muscles. Dermo-lipomata may be associated with accessory auricles and other congenital defects.

Sarcoma is rare. It occurs at the limbus, is usually pigmented, and most of the patients have been old. Sarcomata spread over the surface of the globe, but rarely penetrate it. Recurrence and metastasis occur as elsewhere in the body. They may be alveolar —derived from nævi—or round or spindle celled. They must be removed as freely as possible and examined microscopically. On the slightest sign of recurrence the eye must be excised, and if recurrence again takes place the orbit must be exenterated or

deep X-ray therapy adopted. A diffuse spreading pigmentation of the conjunctiva occurs rarely in elderly people, and has been known to give rise to metastatic sarcomatous tumours. It should always be viewed with grave suspicion.

Epithelioma (*Syn.—Squamous-celled Carcinoma*) occurs par excellence where one kind of epithelium passes into another, hence in the eye chiefly at the limbus (Fig. 114), and at the edges

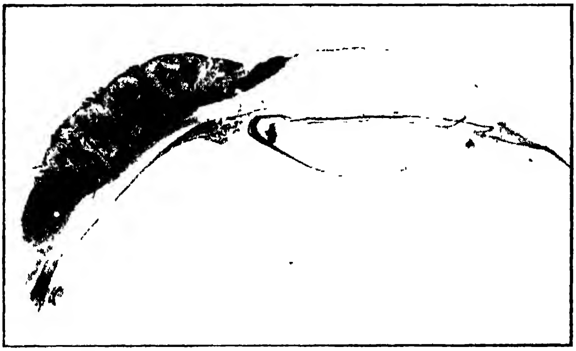


FIG. 114.—Epithelioma of the conjunctiva, from a section (× 6).

of the lids. Papillomata in old people often take on malignant proliferation. Like sarcomata, epitheliomata spread over the surface and into the fornices, rarely penetrating the globe. They have the characteristic structure. The treatment is the same as for sarcoma: it is well in both cases to cauterise the base by diathermy or the actual cautery after the first removal.

Rodent Ulcer (*Syn.—Basal-celled Carcinoma*) may invade the conjunctiva from the lids (*vide* p. 645).

CHAPTER XI

Diseases of the Cornea

THE special importance of diseases of the cornea depends upon the fact that they often leave permanent opacities which seriously lower the visual acuity, while the complications which not infrequently attend them may lead to the loss of the eye.

INFLAMMATION OF THE CORNEA (KERATITIS)

Inflammation of the cornea may be purulent or non-purulent. An immense amount of research has been devoted to inflammation of the cornea from the earliest investigations of Bowman (1849) onwards, and much of our knowledge of inflammation in general is derived from these researches.

Purulent Keratitis, Ulceration of the Cornea. Purulent keratitis is nearly always exogenous, *i.e.*, it is due to pyogenic organisms which invade the cornea from without. The first line of defence is the epithelium. It has been pointed out that the only organisms which are known to be able to invade normal epithelium are the gonococcus and the diphtheria bacillus; but many other bacteria are capable of producing ulceration, notably the pneumococcus.

When we remember the exposed position of the cornea it is not surprising that minute abrasions are extremely common. They are probably of everyday occurrence, but other factors have also to be reckoned with. Pathogenic organisms of high virulence are not always present in the conjunctival sac, and if they are, as is often the case, the resistance of the normal tissues has to be taken into account. It is too often forgotten that normal tissues with a healthy blood supply and lymph flow are well armed against any but the most virulent invaders. Even with these prolonged contact is usually necessary.

Apart from actual abrasions many causes of diminished resistance of the epithelium are met with. Such are drying, as in xerosis, necrosis due to deficient nutrition, as in keratomalacia, desquamation as the result of œdema or neuroparalytic keratitis. If cocaine is instilled too freely, especially

if the lids are not kept closed in the intervals, the epithelium becomes dull and is finally thrown off. Hence this drug is to be used with caution and only under supervision ; it should seldom be given in lotions for use at home.

Malnutrition affects not only the epithelium but also the whole cornea. In badly nourished corneæ ulceration is apt to be deep, leading to early perforation, and also extensive, resulting in widespread necrosis. Such septic ulcers, due to the attack of ordinary pyogenic organisms, occur after injuries, in asthenic conditions, in keratomalacia, lagophthalmia, &c. Corneal ulcers are much commoner in the lower orders, doubtless owing to the defective resistance of the tissues and the more frequent exposure to injury.

In the commonest form of suppurative keratitis—the corneal ulcer—there is localised necrosis in the most anterior layers of the cornea. The sequestrum is partly disintegrated and cast off into the conjunctival sac, and partly adheres to the surface of the ulcer. Usually the epithelium is destroyed and cast off over an area considerably larger than the ulcer itself, and the same applies to Bowman's membrane. The epithelium, however, rapidly advances towards the ulcer, grows over its edge, and even over the slough or pus which forms the floor.

The ulcer is usually saucer-shaped, and the walls project above the normal surface of the cornea (Fig. 115), owing to imbibition of fluid by the corneal lamellæ, which causes them to swell. The spaces between the lamellæ are packed with leucocytes for some distance around the ulcer, appearing as a grey zone of infiltration. This is the progressive stage.

A line of demarcation forms as in necrosis elsewhere in the body. The toxins are most concentrated near the centre where there are most organisms. A wall of polymorphonuclear leucocytes forms a second line of defence. At a certain distance the tissues are protected ; here the leucocytes are not paralysed or killed by toxins, but exert their digestive functions, macerating and dissolving the necrotic tissues. When the dead material has been thrown off the ulcer is somewhat larger, but the cloudiness has disappeared, the floor and edges are smooth and transparent, and the regressive stage is reached.

Meanwhile vascularisation has been going on (Plate V., Fig. 1). Minute superficial vessels grow in from the limbus near the ulcer. They supply the pabulum to restore the loss of substance : they also supply protective substances—anti-

bodies—and therefore play an important *rôle* in combating bacterial infection. Sometimes they are so exuberant as to overstep the limits of utility, *e.g.*, in fascicular ulcer (*vide* p. 221).

When the ulcer has become vascularised, everything is prepared for cicatrisation, which is carried out exactly as in other connective tissues. The fixed connective tissue cells, here the corneal corpuscles, divide and form masses of nucleated spindle-shaped cells, over which the epithelium grows and is lifted to its normal level. The nuclei and vessels gradually disappear, and a mass of fibrous tissue is formed. The fibres are not arranged regularly like the normal lamellæ, so that they refract the light in various directions: the scar is, therefore, more or less opaque according to its thickness. If it is very large and dense some of the larger vessels persist permanently; the smaller ones disappear. Bowman's mem-

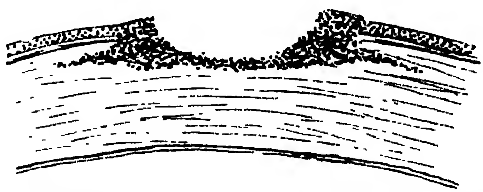


FIG. 115.—Vertical section of a corneal ulcer, showing infiltration of the substantia propria.

brane is never regenerated, and if it has been destroyed, as is the case in all but very superficial abrasions, some degree of permanent opacity remains.

During the progressive stage there is lacrymation, photophobia, and pain, owing to the exposure of the fibrils of the ophthalmic division of the fifth nerve. Some of the toxins elaborated by the bacteria diffuse through the cornea into the anterior chamber, just as atropine does when instilled into the conjunctival sac. Here they exert an irritative effect upon the vessels of the iris and ciliary body, so that hyperæmia of the iris, with or without definite ciliary injection, occurs. The irritation may be so great that leucocytosis takes place, and polymorphonuclear leucocytes are poured out by the vessels of the iris and ciliary processes. They pass into the aqueous and gravitate to the bottom of the anterior chamber, where they form an *hypopyon* (Plate VI, Fig. 2; Fig. 131).

There are several important facts about hypopyon which must be borne in mind. The pus cells do not come from the

cornea, as was once thought. This is proved by the following facts: (1) Descemet's membrane is impermeable to leucocytes, though fluids readily pass through it; (2) the cells sometimes contain pigment granules, obviously derived from the uveal tract; (3) if the ulcer has not perforated, the hypopyon is sterile. The last fact is of the greatest importance, both theoretically and practically. It shows that the leucocytosis is due to toxins, not to actual invasion of bacteria, which, indeed, are as incapable of passing through the intact Descemet's membrane as are leucocytes. It accounts for the ease and rapidity with which hypopyon is often absorbed: it may develop in an hour or two, rapidly disappear, and as readily reappear. Such hypopyons are very fluid, always moving to the lowest part of the anterior chamber if the position of the patient's head is changed. The fact that the hypopyon is sterile has great practical importance—it is unnecessary to remove the pus, as is the rule in all other parts of the body; if the ulcerative process can be stopped the hypopyon will be absorbed.

The hypopyon may be so small that it is scarcely visible, being hidden behind the rim of sclera which overlaps the cornea. It may reach half-way up the iris, having a flat upper surface in accordance with the laws of gravity. It may fill the anterior chamber, wholly obscuring the iris. The larger hypopyons are usually less fluid, owing to the formation of a fibrinous network which imprisons the leucocytes in its meshes. Such hypopyons are much less readily absorbed, and it may be necessary or advisable to evacuate them.

The scar tissue which replaces the destroyed portions of the cornea usually fills in the gap exactly, so that the surface is level. If it is thin the resulting opacity is slight and is called a *nebula* (Plate V., Fig. 1); if rather more dense it is sometimes called a *macula;* if very dense and white it is called a *leucoma.* Old central leucomata sometimes show a horizontal pigmented line in the palpebral aperture, the nature of which is obscure. A thin, diffuse nebula covering the pupillary area interferes more with vision than a strictly localised dense leucoma, so long as the latter does not block the whole pupillary area. The reason is that the leucoma stops all the light which falls upon it (Fig. 116), whereas the nebula refracts it irregularly, allowing many of the rays to fall upon the retina, where they blur the image formed by the regularly refracted rays. An opacity does not necessarily prevent the light from being focussed upon the retina immediately behind it. Thus,

PLATE V.

FIG. 1.—Nebulæ.

FIG. 2.—Phlyctenular pannus.

[*To face p.* 200.

PLATE VII.

FIG. 1.—Interstitial keratitis.

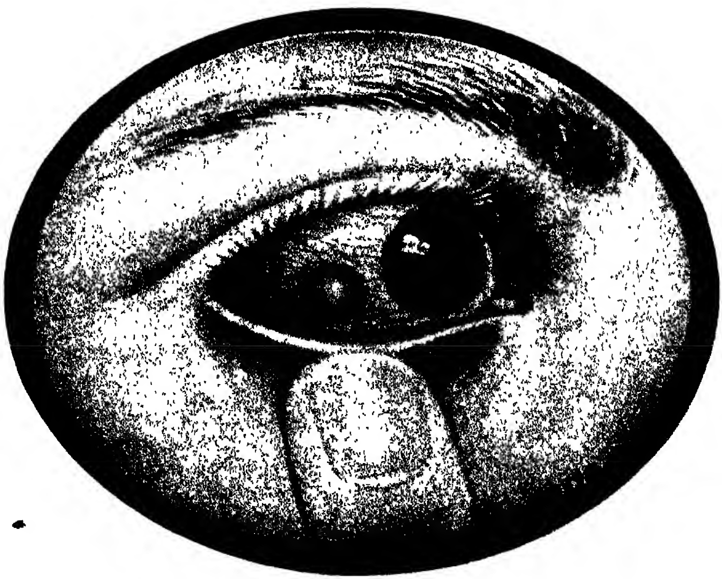


FIG. 2.—Episcleritis.

a central opacity of the cornea will not prevent the focussing of an object upon the macular region, for the rays passing through the clear peripheral parts of the cornea will be refracted towards the macula, only those rays being cut off which are incident to the corneal surface at the opaque region. There is thus a loss of brightness rather than of definition, though definition will also be impaired by the superposition of a diffuse entoptic image of the opacity upon the clear image of the external object.

When Bowman's membrane has been destroyed the opacity is permanent, but even then it tends to clear more or less. The younger the patient the more clearing may be anticipated. The deeper the cicatrix the less it clears; perforating wounds remain permanently opaque. This fact is well illustrated by

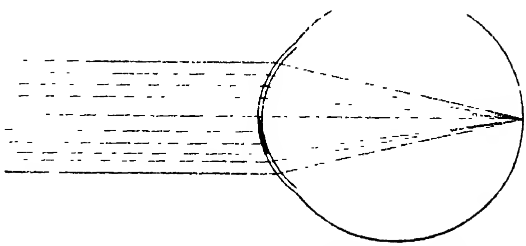


Fig. 116.—Optical effect of a corneal opacity.

the punctures made by discission needles in needling cataract; they remain as grey spots in the cornea throughout life. Vascularisation plays a part in the clearing of corneal opacities, as is shown by the fact that they clear first in the immediate vicinity of the vessels.

Extremely thin cicatrices may be almost or quite transparent. In them there is often deficient scar tissue formed so that the surface is flattened or even indented. Such corneal *facets* can only be seen by carefully examining the corneal reflex (*vide* p. 86), but they cause considerable defect of visual acuity.

From the same cause—deficiency of scar tissue—the cornea may be markedly thinner at the site of the ulcer than elsewhere. The scar may then bulge above the surface, owing to ineffectual resistance to the normal intraocular pressure. As the cicatrix becomes consolidated the bulging may disappear, or it may remain permanently as an *ectatic cicatrix* (keratectasia from ulcer).

Some ulcers, especially those due to the pneumococcus and

septic organisms, extend rapidly in depth. There is then grave danger of perforation. The whole thickness of the cornea except Descemet's membrane and a few corneal lamellæ may be destroyed. Descemet's membrane, like other elastic membranes, offers great resistance to inflammatory processes. It is, however, unable alone to support the intraocular pressure: it therefore becomes protruded through the ulcer, appearing upon the surface as a transparent vesicle, which is called a *keratocele*. This may persist, surrounded by a white cicatricial ring, or it may eventually rupture.

Perforation and its effects. When an ulcer perforates the aqueous suddenly escapes and the intraocular pressure sinks to zero, *i.e.*, to the atmospheric pressure. The iris and lens are driven forwards into contact with the back of the cornea.

The effect of perforation upon the nutrition of the cornea is good: owing to the diminution of intraocular pressure the diffusion of lymph through the cornea is facilitated. Ulceration usually ceases, pain is alleviated, and cicatrisation proceeds rapidly. The complications which attend perforation are, however, of extreme danger to sight and even to the preservation of the eye. These complications vary according to the position and size of the perforation.

Usually the perforation takes place opposite some part of the iris, which therefore lines the aperture when the aqueous escapes. The iris becomes gummed down to the opening by lymph, which gradually organises, and an *anterior synechia* is formed. The blocking of the perforation with iris allows the anterior chamber to be re-formed, fresh aqueous being rapidly secreted.

The aqueous often escapes very quickly owing to some sudden exertion on the part of the patient, *e.g.*, coughing, sneezing, straining at stool, or spasm of the orbicularis. Any such sudden exertion causes a rise in general blood pressure, which at once manifests itself by a rise in intraocular pressure. The weak floor of the ulcer is unable to support the sudden strain and gives way. In such a case, especially if the perforation is large, a portion of the iris is carried not only into the opening but through it, and a *prolapse of iris* is produced. The prolapse may not include the pupillary margin, in which case it is hemispherical; or the pupillary margin may also prolapse, a tag of iris lying free upon the cornea. In either case the colour of the iris soon becomes obscured by the deposition of grey or yellow lymph upon the surface. In large

prolapses the stroma becomes thinned and the black retinal pigment epithelium is thrown into relief.

Sometimes the whole cornea sloughs, with the exception of a narrow rim at the margin, and *total prolapse of iris* occurs. The pupil usually becomes blocked with exudate, and a false cornea is formed, consisting of iris covered by lymph.

If the perforation takes place very suddenly the suspensory ligament of the lens is much stretched. It may rupture partially, causing tilting or dislocation of the lens, or wholly, so that the lens may be expelled through the perforation.

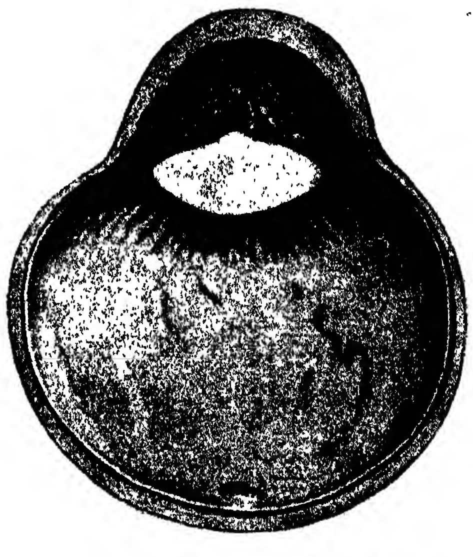


FIG. 117.—Anterior staphyloma, showing also an anterior capsular (pyramidal) cataract. (R. L. O. H. Museum.)

If prolapse of iris has occurred cicatrisation may still progress. The lymph which covers the prolapse or pseudo-cornea becomes organised, and forms a thin layer of connective tissue over which the conjunctival or corneal epithelium rapidly grows. The contraction of the bands of fibrous tissue tends to flatten the protruding prolapse or pseudo-cornea. It rarely, however, becomes quite flat; more commonly the iris and cicatricial tissue are too weak to support the restored intraocular pressure, which is often increased, owing to the advance in position of the iris (*vide* p. 281). The cicatrix tends therefore to become ectatic, and such an ectatic cicatrix in which the iris is incarcerated is called an *anterior staphyloma* (Fig. 117). If the prolapse of iris is partial the resulting staphyloma will be partial; if total, a total staphyloma will ensue. The bands of scar tissue on the staphyloma vary in breadth and thickness, producing a lobulated surface, hence the name (σταφυλή, a bunch of grapes).

If the perforation happens to be opposite the pupil it cannot be covered with iris. The pupil often becomes adherent to the margin, and the aperture becomes filled with exudate. The anterior chamber is then re-formed very slowly: the lens remains long in contact with the ulcer, and permanent opacity may occur in it—*anterior capsular cataract* (Fig. 117). As the anterior chamber re-forms the exudate filling the opening is submitted to strain. It frequently ruptures, especially if the patient is restless. This process may be repeated again and again, so that the opening may become permanent—*corneal fistula*.

The sudden reduction of intraocular pressure when the perforation occurs removes support from all the intraocular blood vessels. They become dilated and may rupture, *intraocular hæmorrhage* taking place. The retinal vessels may rupture, causing vitreous hæmorrhage, or the choroidal, causing sub-retinal or sub-choroidal hæmorrhage. It may be so profuse that the contents of the globe are extruded with the outflowing blood; indeed, in very rare cases the hæmorrhage may endanger life, for it is most likely to occur in old people with atheromatous vessels.

Finally, the organisms which have caused the ulceration of the cornea may gain access to the interior of the eye as the result of perforation, the vitreous acting as an excellent culture medium. *Purulent irido-cyclitis* or even *panophthalmitis* may thus be set up, a result especially prone to occur in gonorrhœal ophthalmia and in hypopyon ulcer (*q.v.*).

Treatment of Uncomplicated Ulcers. Control of infection, cleanliness, heat, rest, and protection are the fundamental principles of the treatment of corneal ulcers. Control of infection is attained by the use of bacteriostatic drugs: surgical cleanliness is the principle which should regulate the use of lotions; heat is employed to prevent stasis and encourage repair; local rest is attained by the use of atropine; rest and protection from deleterious external agencies are aimed at in the use of a pad and bandage.

The ordinary treatment of a simple uncomplicated ulcer is as follows: The conjunctival sac is washed out carefully three or more times a day with a considerable quantity of a mild antiseptic lotion, which should be used as hot as can be borne comfortably (*vide* p. 182). It acts principally by washing away secretions and necrotic material, which carry with them many of the organisms and their toxins. It is impossible to apply

antiseptics sufficiently strong to kill the organisms, hence it is of little importance whether weak sublimate lotion (1 in 8000) or simple boric lotion be used.

The infection is controlled by the local use of bacteriostatic drugs such as penicillin solution (1000 to 2000 units per c.c.) as drops repeated every quarter of an hour for the first two hours and then periodically as indicated (*vide* p. 696). In a mild case three or four instillations will suffice. Before each the eye should be irrigated and a drop of 1 per cent. atropine solution or a small lump of 1 per cent. atropine ointment introduced between the lids.

A protective pad and bandage are then applied. It consists of a pad of sterile gamgee tissue, or a layer of cyanide gauze covered by a pad of cotton wool, kept in place by a bandage, firmly but securely applied. A simple tie bandage suffices. This is of sufficient length to pass round the head and tie behind. It passes obliquely above the sound eye, over the ulcerated eye, and under the ear of the side of the affected eye, where the bandage is given a single turn; the ends are tied just above the occipital protuberance.

This treatment suffices for mild cases.

In more severe cases the penicillin treatment should be intensified and hot bathings or compresses (*vide* p. 694) should be applied in the intervals. The compresses should be made of large round pads of plain or boric lint, on one surface of which gutta-percha tissue is sewn. The compresses are placed in a cloth and immersed in boiling water; by keeping the ends of the cloth out of the water and turning them in opposite directions the excess of the water is wrung out without scalding the fingers. The compress is applied as hot as can be borne. It is at once covered with a large pad of hot cotton wool, and bandaged into position.

Atropine has a twofold function in the treatment of corneal ulcers. In the first place it keeps the eye at rest by paralysing the intrinsic muscles, both the sphincter iridis and ciliary muscle. In the second place it prevents most of the dangerous results of iritis (*vide* p. 259). Corneal ulcers are always accompanied by more or less iridic and ciliary hyperæmia, and actual inflammation often occurs.

Eserine has been recommended instead of atropine in the treatment of some ulcers. It has been held that prolapse is less likely to occur when a peripheral ulcer perforates if the pupil is contracted. This is a fallacy. Even with complete dilatation under atropine the pupil at once contracts when the

aqueous escapes. The objections to eserine are that it prevents rest by keeping the sphincter iridis and ciliary muscle in a state of tonic contraction, that it irritates the iris and tends to increase iritis, with consequently greater risk of the formation of posterior synechiæ, and that it causes discomfort or even pain in the eye. It is never to be used for a simple ulcer, though it may have beneficial effects in special chronic types (*vide* p. 223).

Any contributory cause for ulceration must of course receive attention. Prominent among such causes are conjunctival conditions and general malnutrition. Thus, the ulcers asso-

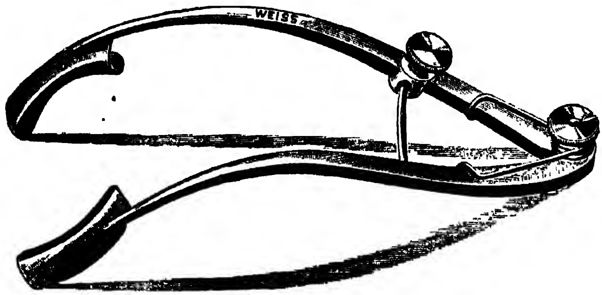


FIG. 118.—Lang's speculum. This form is preferable to the ordinary type, as it keeps the lashes out of the field of operation.

ciated with trachomatous pannus will not heal if the lids are neglected (*vide* p. 179).

In purulent conjunctivitis and trachoma the lids should be painted with silver nitrate, even during the progressive stage. The copper stick must not be used in trachoma, owing to the irritative effect upon the cornea. The presence of much conjunctival discharge is a contraindication to the use of a bandage; the benefit derived from it is more than counteracted by the retention of secretions (*vide* p. 153). It must be replaced by a shade.

To restrain children from touching their bandages a cylinder of stout corrugated cardboard may be applied around the arm, reaching a little beyond the elbow, thus preventing flexion.

In debilitated adults or old people and marasmic children the building up of the constitution by good food, fresh air, and tonics is often more important than the treatment of the local

condition. Large doses of vitamin C, injected intravenously if necessary, are frequently of considerable value in cases where the general health is not good.

When cicatrisation is complete and all irritative signs have passed off an attempt must be made to render the scar as transparent as possible. The results are usually disappointing, but cicatrices clear considerably in young patients, and in many others a gratifying improvement may be noticed in the course of months or years. Stimulating treatment is indicated, beginning with weak irritants and passing cautiously to stronger. Insufflations of finely powdered calomel may be used first. If it is well borne dilute yellow oxide of mercury ointment is employed. A small lump of the ointment, gr. iv. to ℥ i., is placed in the conjunctival sac and rubbed in gently by rotatory movements of the upper lid by means of the finger. The massage with the lid should be employed three times a day for periods of 5 to 10 minutes. If it is not resented stronger ointment, up to gr. xvi. to ℥ i., is used. As the eye becomes accustomed to one form of irritation it is well to change the drug from time to time. The use of 2 per cent. quinine bisulphate ointment twice a day has been advocated. Dionin, 5 to 10 per cent., may be added to the ointment, or used in solution. On application, especially for the first few times, it causes great œdema of the conjunctiva and a burning sensation. The surgeon should make the first application, as the patient is often alarmed at its severity. The stimulation of the blood and lymph flow induced by this drug is undoubtedly beneficial.

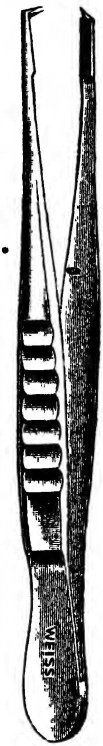
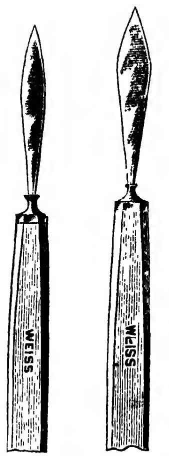


FIG. 119.—Fixation forceps with "blocked" ends for holding a suture needle.

In very intractable cases the same effect may be produced by subconjunctival injections of sodium chloride solution (2 to 10 per cent.) or oxycyanide of mercury (1 in 5,000) after instillation of cocaine. Five to ten minims of sterile solution are injected under the bulbar conjunctiva as far as

possible behind the upper part of the limbus. The pain, which may be severe, is diminished by the addition of 2 per cent. novocain to the solution. The injection should not be repeated more than once a week.



FULL SIZE FULL SIZE
FIG. 120. FIG. 121.
Broad needles.

Old, degenerated, often calcareous cicatrices are best left alone, since they are liable to break down and form very dangerous ulcers, owing to the lack of resistance in the scar tissue.

In some cases of superficial keratitis the scar remains permanently richly vascularised. These eyes are often extremely irritable and give rise to repeated attacks of inflammation and lacrymation, which may make life a burden. Though uncertain in its results, peritomy (*vide* p. 182) is the best treatment. The re-growth of large new vessels must be prevented; it is best effected by painting the new vessels with a very fine camel's hair brush which has been moistened and then rubbed on a silver nitrate stick, or by touching them with a diathermy needle.

If a small dense leucoma covers the pupillary area vision may be improved by an optical iridectomy (Chap. XXII.).

Keratoplasty, the excision of a disc of scarred cornea. and its replacement by a disc of clear cornea from a human eye, is sometimes disappointing, and is only to be resorted to if blindness is otherwise inevitable.

(3) Some improvement in appearance may be obtained by tattooing dense leucomata. It is only suitable for firm smooth scars in perfectly quiet eyes, and is even then not without danger. More justifiable is the tattooing of small central nebulæ; it has the effect of cutting off the irregularly refracted rays, so that vision is improved (*vide* p. 201). Tattooing with Indian ink has been replaced by impregnation with gold (brown) or platinum (black): of these the latter is preferable. The required area is denuded of epithelium and a piece of blotting-paper of the same size, soaked in platinum chloride solution, is applied. On removal a few drops of fresh hydrazine hydrate are allowed to flow over the area, which becomes black. The eye is irrigated

with saline, a drop of parolein instilled, and a pad and bandage put on.

Treatment of Complicated Ulcers. If perforation is imminent special means must be adopted to prevent it. The patient should be confined to bed, and laxatives given.

It has already been pointed out that perforation improves the nutrition of the cornea. Perforation may sometimes be anticipated with advantage by *paracentesis*. By this procedure the aqueous is evacuated slowly, and the more dangerous

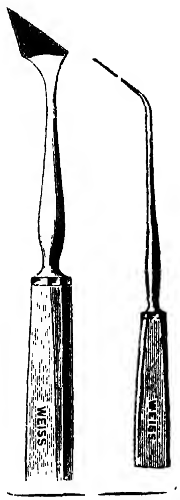


Fig. 122.—Keratome.

Fig. 123.—Desmarres' paracentesis needle.

Fig. 124.—Spatula.

results of perforation may be avoided. Another indication for paracentesis is extreme pain.

Paracentesis may be performed through the floor of the ulcer or just inside the periphery of the cornea. When the aqueous has escaped and the inflamed iris comes in contact with the cornea the most acute pain is felt. The eye should therefore be anæsthetised by injection of novocain into the orbit or into Tenon's capsule (*vide* p. 471).

The patient lies upon his back upon the operating table. The conjunctival sac is washed out with warm boric lotion

or saline. The speculum (Fig. 118) is inserted, and the eye is fixed with fixation forceps (Fig. 119) by taking up a fold of conjunctiva at a convenient spot close to the corneal margin. The points of the forceps should be pressed well into the conjunctiva so as to include the episcleral tissue, otherwise they are liable to tear the conjunctiva if much traction is exerted, as by an involuntary movement of the patient. The eye is then pulled gently forwards. The incision is made with a broad needle (Figs. 120, 121), or a keratome, preferably one bent on the flat (Fig. 122), or a paracentesis needle (Fig. 123). If the floor of the ulcer is to be incised the point is inserted here so that the blade makes an angle of about 45° with the cornea. Directly it is pushed through the floor the plane of the blade is altered so that it lies against the back of the cornea; if this is not done there is imminent danger of wounding the lens. The instrument is pushed on until the incision is sufficiently long. It is then very slowly withdrawn, so that the aqueous may flow off very gradually. If the aqueous escapes suddenly the lens may be wounded, intraocular hæmorrhage may occur, or the iris may prolapse. If the last misfortune occur the prolapse must be excised (*vide* p. 212). If the operation is performed well, probably little aqueous will escape. The spatula (Fig. 124) is then applied to the edge of the wound which is nearer to the corneal margin, and this lip is gently depressed. The aqueous then escapes slowly and with a minimum disturbance to the eye. The aqueous can be evacuated on the following day by simply opening the wound with the spatula and depressing the lip.

If the incision is made near the periphery of the cornea it should be 1 to 2 mm. inside the lower margin, especially if there is an hypopyon present. The keratome is then entered in the plane of the iris, and its direction changed as before as soon as the point is seen to be inside the anterior chamber.

In deep ulcers, such as are liable to perforate, the removal of necrotic material may be hastened by scraping the floor with a spatula, or the ulcer may be cauterised (*vide* p. 217). If the actual cautery is used it may be made deliberately to perforate the centre of the floor of the ulcer, so that the aqueous may escape and better conditions of nutrition be set up. This procedure can only be recommended in special cases.

Another procedure is to scrape the floor of the ulcer and

then to cover it with a flap of conjunctiva. The conjunctiva is separated from the limbus near the ulcer. A second incision is made with scissors parallel to the first, so that a strip of conjunctiva rather wider than the breadth of the ulcer is separated from the globe but remains attached at the

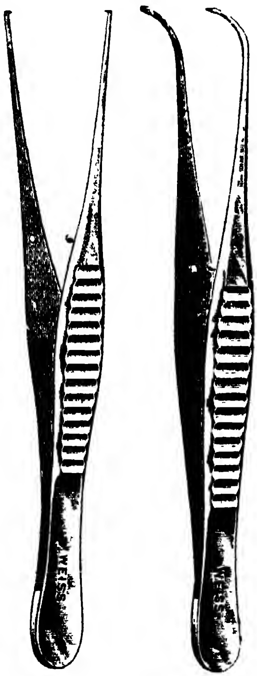


Fig. 125. Fig. 126. Iris forceps, straight and bent.

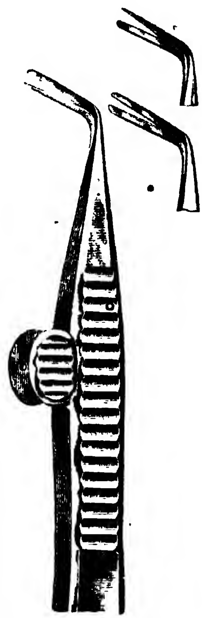


Fig. 127. — de Wecker's scissors. They should be blunt-pointed for iridectomy, one blade sharp-pointed for iridotomy.

two ends. This is then shifted on to the cornea so as to cover the ulcer, being retained in place by a stitch at one or both ends, thus keeping it somewhat stretched. The wound in the bulbar conjunctiva may be closed by a stitch. The lids are then carefully lifted over the strip, so as not to displace it, and the eye is bandaged.

If perforation has occurred the treatment depends upon its size and situation. If it is small or in the pupillary area prolapse of iris is not to be feared. Rest in bed, atropine, and a firmly applied bandage suffice: all forced expiration—blowing the nose, coughing, &c.—must be avoided. A sneeze can often be inhibited by firm pressure with the finger upon the middle of the upper lip close to the nose. If a small perforation is over the iris, adhesion to the cornea usually occurs. It may become detached when the anterior chamber re-forms, or may be drawn out into a fine thread. No special treatment is required.

If prolapse of iris has occurred it should usually be excised (*vide infra*). No attempt should be made to replace the prolapse in these cases because the iris has become soiled with pus, and replacement may result in infection of the interior of the eye and panophthalmitis.

Iridectomy of prolapsed iris is performed as follows: Instruments required: speculum, fixation forceps, two pairs of iris forceps (Figs. 125, 126), de Wecker's scissors (Fig. 127), iris repositor (Fig. 128).

If the patient is a child or highly excitable, a general anæsthesia is used, otherwise local anæsthesia, with or without novocain, suffices (*vide* p. 201). The conjunctival sac is washed out (*vide* p. 152). The speculum is inserted, and the eye fixed with fixation forceps held in the left hand. The iris repositor is passed into the wound between the iris and the cornea, with a view to freeing any adhesion. The longer the prolapse has existed the firmer will be the adhesion of the iris to the cornea. It may be impossible to free it, and in this case effectual iridectomy cannot be performed. Having freed the iris as much as possible, the fixation forceps are handed over to an assistant. The prolapse is seized with iris forceps held in the right hand, as close to the cornea as possible, and drawn well out from the wound. The second pair of iris forceps, held in the left hand, is then applied again as near the cornea as possible, and the iris drawn still further out. (Capsule forceps (Fig. 129) are very good for this purpose.) de Wecker's scissors are then taken in the right hand, and

FIG. 128.—Iris repositor, made of silver; it can be bent to any required angle.

the iris is cut off close to the cornea. If the operation has been successfully performed the stump of iris retracts into the anterior chamber and is quite free from the wound. Atropine is instilled, and a pad and bandage applied.

It is to be noted here that the iris is extremely ductile; it can be dragged out much farther than might be expected, and it must be dragged out as far as possible in order that the incision may be through clean iris tissue, all the soiled part being removed. Some operators prefer to retain the fixation forceps in the left hand throughout, drawing out the iris with iris forceps in the right hand. In this case the assistant cuts off the prolapse. The method has the advantage that any sudden movement of the eye may be counteracted or followed by co-ordinated movements of the two hands of the same individual. Such sudden jerks have been known to drag the whole iris out of the wound, since it tears away at the thinnest part, viz., near the ciliary border. In ordinary cases the greatest danger is that of wounding the lens. A conjunctival flap may be used after excision of the prolapse (*vide* p. 211).

Iridectomy of prolapsed iris is only possible within the first few days, before adhesion has become firm. It is not to be performed after this has occurred, nor in the case of very large prolapses. In the latter there is so large an opening in the cornea that a permanent fistula may result, with loss of the eye from diminished tension and shrinking.

In very large prolapses there is much bulging and the base is often constricted. Every attempt should then be directed to obtain a flat cicatrix. In addition to rest in bed and the means already advised, a pressure bandage must be applied for a prolonged period. A pressure bandage differs from an ordinary protective bandage only in that the space around the eye is packed carefully with cotton wool to the level of the nose and that considerable pressure is exerted in applying the bandage.

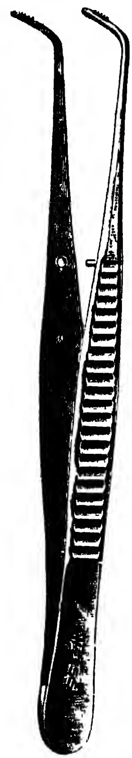


FIG. 129. — Couper's capsule forceps.

Keratocele is treated first by rest and a pressure bandage. If this fails the vesicle may be punctured, and the case treated like a perforated ulcer.

Fistula of the cornea is treated first like a perforated ulcer. If this treatment fails the edges of the fistula may be cauterised with the actual cautery, or a point of lunar caustic. In order that this may be done there must be some trace of an anterior chamber, otherwise the lens will be injured. A conjunctival flap may be drawn over the fistula.

Commencing staphyloma should be treated by a pressure bandage. If this fails a paracentesis may be done, or an iridectomy opposite the clearest part of the cornea.

Hypopyon Ulcer. When an eye is injured so that an abrasion of the cornea is produced there is grave danger of infection from virulent pyogenic organisms. The probabilities of this occurrence depend upon the presence of such organisms and upon the amount of resistance which the tissues possess. Of all the organisms which are capable of producing deep ulceration by far the most dangerous, because the most widely spread, is the pneumococcus. It is not infrequently present in the normal conjunctival sac, but it is particularly likely to be present if there is any inflammation of the lacrymal sac (dacryocystitis). The presence of dacryocystitis is therefore a standing menace to the eye. The pneumococcus, more than any other organism, tends to give rise to hypopyon, but other pyogenic organisms may also produce this result.

The substance which produces the injury may carry the infecting agent. The commonest causes are scratches with the finger nail, leaves or branches, grains of corn, and minute foreign bodies, especially stone.

Unless the organism be very virulent some lack of resistance on the part of the tissues must be predicated. Hence hypopyon ulcers are much commonest in old people and alcoholic subjects, and in the lower rather than the upper classes. The debilitating effects of hot weather are noticeable. Hypopyon ulcers also occur during or after acute infectious diseases, such as measles, scarlet fever, small-pox, vaccinia and so on. In small-pox it is not a variolous pustule upon the cornea, but it differs from the typical hypopyon ulcer.

Hypopyon ulcers vary in type according to the infective agent and the age of the patient. In about 70—80 per cent. of all cases in adults the cause is the pneumococcus, and the ulcer is then of a very characteristic type, and has been called *ulcus serpens* from its tendency to travel over the cornea in a serpiginous fashion.

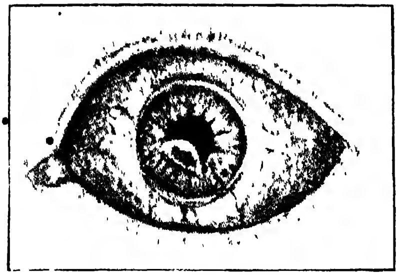


FIG. 1.

Fig. 1.—Ulcus serpens, with crescentic infiltrated advancing border above. There was no hypopyon when the figure was drawn. (From a drawing by Dr. S. H. Habershon.)

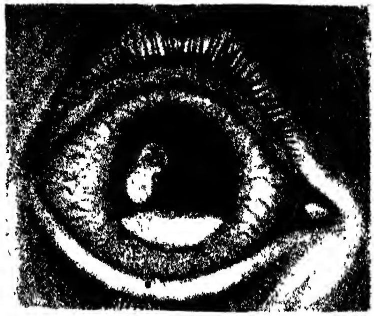


FIG. 2.

Fig. 2.—Hypopyon ulcer.

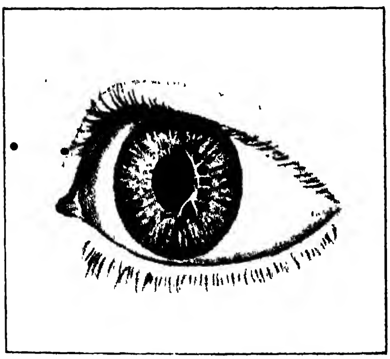


FIG. 3.

Fig. 3.—Persistent pupillary membrane. Note the origin of the strands from the position of the minor arterial circle.

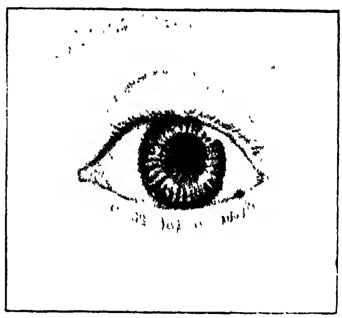


FIG. 4.

Fig. 4.—Iritis, with irregular pupil and ring synechia, causing bulging forwards of the iris ("iris bombé").

[*To face p.* 214.

The typical ulcus serpens is a greyish-white or yellowish disc near the centre of the cornea (Plate VI., Fig. 1). The opacity is greater at the edges than at the centre and is particularly well marked in one special direction. A cloudy grey area, made up of fine lines, surrounds the disc, but is also more marked in the same direction. The whole of the cornea may be lustreless or hazy. There is a violent iritis, and the aqueous is cloudy, or there may be a definite hypopyon. The lids are slightly œdematous, and there is conjunctival and ciliary congestion. The subjective symptoms at the early stage are pain in the eye and brow and a variable amount of photophobia. There is remarkably little pain after the initial stage; hence treatment is often unduly delayed, with disastrous results.

The ulcer increases in size and depth. On the side of the densest infiltration, which often looks like a yellow crescent, the tissues break down and the ulcer spreads; on the other side it may be undergoing simultaneous cicatrisation. In this manner it travels forwards. Meanwhile the hypopyon has become more evident, but it may vary in size from hour to hour (*vide* p. 200). As might be expected, the intraocular pressure is often raised.

If left to pursue its natural course the hypopyon will increase and become fibrinous, the ulcer will perforate, usually forming a large opening through which the iris prolapses. The whole cornea, except the narrow rim nourished by the corneal loops (Plate II.), may necrose, and panophthalmitis destroy the eye. In other cases an extremely dense cicatrix in which the iris is incarcerated (*adherent leucoma*) destroys sight. This may be flat or ectatic (anterior staphyloma). Sometimes the iris is bound down to the lens before perforation occurs. In such cases there are posterior synechiæ, which may be annular or total (*vide* p. 260), and the pupil may be blocked by exudates which organise into fibrous tissue (occlusion of the pupil).

Though hypopyon ulcer occurs sometimes in children, it never assumes the typical form of an ulcus serpens. In them and in some cases in adults the serpiginous character of the ulcer is not apparent, but it is distinguished by its great tendency to extend in depth, so that perforation readily occurs. On the whole, such ulcers have a milder course than the ulcus serpens, and this is especially the case in children. This is doubtless due partly to the fact that less virulent organisms are at work, especially applicable to adult cases, and partly

to greater resistance of the tissues, especially applicable to children.

The milder type of hypopyon ulcer is often due to the diplobacillus of Morax or to the allied diplobacillus liquefaciens of Petit. It usually commences as a central grey infiltration, which develops into an ulcer covered by a grey membrane and surrounded by radiating grey striæ. It generally spreads in all directions, but does not show the same tendency as the pneumococcic ulcer to spread in depth. It can only be diagnosed with certainty by bacteriological examination.

Pathology. The ulcus serpens is due to the pneumococcus, either alone or mixed with other organisms. There is no doubt that the essential features are caused by the pneumo-

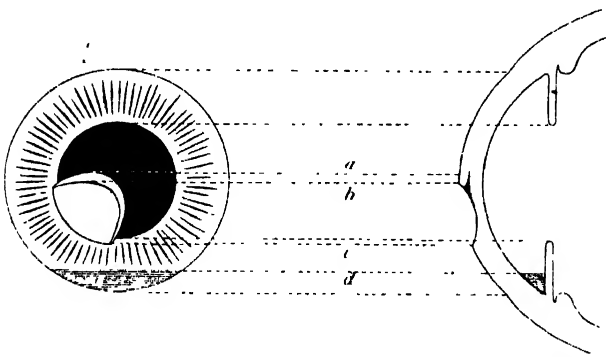


FIG. 130.—Diagram of hypopyon ulcer *a—b*, advancing infiltrated border; *b—c*, ulcerated surface; *d*, upper level of hypopyon.

coccus alone. A variety of organisms—staphylococci, streptococci, gonococci, &c.—have been found in atypical hypopyon ulcers. As already stated, a relatively mild form is due to the diplobacillus.

Anatomically, the ulcus serpens shows at first a depressed surface covered with slough (Fig. 130). The corneal lamellæ around and below the ulcer are separated by masses of polymorphonuclear leucocytes.

In the progressive stage the infiltration is chiefly limited to an area, wedge-shaped in section, corresponding with the yellow crescent. In other parts the edges are clean, and may be covered with epithelium. Often there is infiltration just anterior to Descemet's membrane at a spot exactly opposite the floor of the ulcer, while the intervening lamellæ are normal. This fact accounts to some extent for the great tendency to

perforation, since the inflammatory process is going on as it were from both surfaces of the cornea.

The hypopyon consists of polymorphonuclear leucocytes massed together in the lower angle of the anterior chamber. In the later stages they are enmeshed in a network of fibrin. It has been pointed out that the leucocytes are derived from the iris and ciliary processes (*vide* p. 199).

Treatment. In all cases of hypopyon ulcer in adults treatment must be initiated at once and must be energetic. An initial attack should be made with penicillin, a solution (1,000 to 2,500 units per c.c.) being dropped on to the cornea or, in the first place, a few crystals of the salt applied to the surface of the ulcer itself for approximately a minute while the eyelids are held apart. This should be done after the overhanging edge and the underlying infiltrated tissue have been curetted with a small spud. An alternative and very effective method is the subconjunctival injection of penicillin: if the pure salt is available doses of the order of 10,000 units can be repeated every three hours. The bacteriology should be investigated and if the organisms are found to be insensitive (which is rare) the treatment can be discontinued, but penicillin treatment should not be delayed until the cultures are developed.

If the response to penicillin is not good, or if the organisms are not responsive to this drug, a full and intensive course of one of the sulphonamides should be given (*vide* p. 695); albucid drops applied locally have given good results. Atropine is instilled even if the tension is raised (*vide infra*).

Coincidentally with this treatment the ulcer may be cauterised. If this is performed skilfully it does no harm and may save the eye. It is seldom necessary in children.

Cauterisation may be performed with pure carbolic acid or trichloracetic acid (10-20 per cent.) or with the actual cautery, the most convenient form of the latter being the galvanocautery (Fig. 131). In my opinion the latter method, which requires more skill, possesses no advantages and has some disadvantages. Apart from the dangers attending the use of the actual cautery, carbolic acid has the advantage of penetrating a little more deeply than it is actually applied, thus extending its antiseptic properties more widely; it acts both as a caustic and an antiseptic. No harm is done even if the acid spreads over the normal cornea. Although the parts touched become at once quite white, the normal tissues rapidly recover without detriment. The acid must not, however, touch the

conjunctiva, otherwise very acute conjunctivitis is set up and adhesion between the lids and globe may occur.

Pure carbolic acid is applied as follows: The patient is seated or lying upon a couch. The ulcer is first stained with fluorescein (2 per cent.), in order that its limits may be more clearly defined. The conjunctival sac is thoroughly anæsthetized (*vide* p. 470). The surgeon stands behind or at the head. With his left hand he separates the lids as in removing a foreign body, steadying the globe at the same time. The ulcer is scraped with a spatula, and together with the surrounding cornea is dried with the point of a piece of blotting paper. A wooden match, somewhat pointed, is dipped into the carbolic acid. Care is taken that the wood is thoroughly wet, but has no drop of acid hanging to it which may run over the cornea. The ulcer is then touched over the whole of its surface with the point of the match. If there is sufficient carbolic acid on the match the spot touched becomes white. Special care is taken

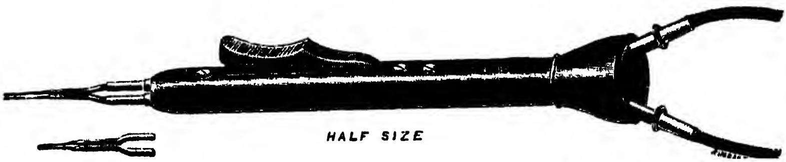


FIG. 131.—Ophthalmic galvano-cautery, which may be worked off the main with a transformer, or off a portable accumulator.

thoroughly to cauterise the advancing edge of an ulcus serpens, *i.e.*, the part marked by a yellow crescent.

Cauterisation with carbolic acid may be repeated two or three times at intervals of one or two days if the ulcer still progresses. If this treatment, combined with the use in the intervals of mild antiseptic lotions, atropine, and hot bathings, as for less severe ulcers, does not check the progress, the actual cautery may be tried. It has been recommended to heat the ulcer with the cautery without actually touching it; or hot air may be blown on to it from a rubber ball, such as is used by dental surgeons. This treatment is certainly beneficial in some cases.

If these means fail, and especially if the tension of the eye is raised (*vide infra*), yet more drastic measures must be resorted to. Of these the most important is *paracentesis*. It may be performed as already described, but in the case of hypopyon ulcers what is called *Saemisch's section* (though it was devised by Guthrie) offers some advantages. It consists

in completely dividing the ulcer from one side to the other, the ends of the incision being in healthy corneal tissue. It must be remembered that if the operation is performed under cocaine the most excruciating pain is felt when the iris comes into contact with the cornea. For this reason it is best to use a general anæsthetic, or infiltrate deeply with novocain.

FIG. 132.—Narrow Graefe knife.

Saemisch's Section. Instruments required: speculum, fixation forceps, Graefe knife (Fig. 132), smooth iris forceps; in case the iris should prolapse, the following should also be at hand: two pairs of iris forceps, de Wecker's scissors, iris repositor.

After anæsthetising, the eye is washed out and the speculum is inserted. The eye is fixed with fixation forceps in the usual manner (*vide* p. 210). The point of the Graefe knife is inserted in healthy cornea just outside the edge of the ulcer, preferably at the advancing part where the yellow crescent is densest. The edge of the knife is directed forwards, so that if the lens advances before the section is complete it will touch the back of the knife and not be wounded. The knife is passed across the anterior chamber until the point is seen beyond the opposite edge of the ulcer. The counter-puncture is made in healthy cornea here, and the knife is pushed on so as to cut out. The aqueous pours out, and carries with it much of the hypopyon. Atropine is instilled, and the eye bandaged.

If the ulcer is very large it is impossible to make the puncture and counter-puncture in healthy cornea. In these cases, too, the knife usually cuts out as it is in the act of passing across the ulcer. If the hypopyon is very fibrinous it may be necessary to pull the coagulum out with the smooth iris forceps.

This procedure improves the conditions of nutrition of the cornea and evacuates the hypopyon. It is not to be advocated in mild or in the early stage of severe cases, but as a last resource it may prove beneficial.

Instead of the ordinary sublimate lotion hydrogen peroxide may be used. Optochin or ethyl hydrocupreine, a quinine derivative, is said to have a specific action on pneumococcic ulcers: a 1 per cent. watery solution of the hydrochloride is

dropped into the eye frequently or it may be applied directly to the ulcer.

The diplobacillary hypopyon ulcer should be treated with zinc salts. Zinc sulphate solution (0·25—1 per cent.) should be used every hour or two, and an ichthyol (1·5 per cent.)-zinc sulphate (0·5 per cent.) ointment applied to the lids.

The results of treatment of the severer forms of hypopyon ulcer are disappointing. This is largely due to the fact that they are seen too late. In rabbits it is possible to control the development of pneumococcic ulcers by intravenous injection of an anti-pneumococcic serum, if this be used sufficiently early. This method has been adopted in man, but usually fails. *The commonest cause of failure is the development of secondary glaucoma.* The patients are usually elderly and therefore often have shallow anterior chambers. In the absence of an ulcer one would hesitate to put atropine into such an eye from the dread of causing glaucoma (*vide* p. 285). Now the presence of an ulcer, with the accompanying iritis and hypopyon, increases the risks of glaucoma from the use of atropine. We are therefore on the horns of a dilemma, for atropine will have a beneficial effect in keeping the iris at rest and tending to diminish the iritis, and therewith the hypopyon. The routine use of atropine is therefore justified, but the tension of the eye should be watched much more carefully than is usually done. If the tension rises the effect on the cornea is extremely bad, for it diminishes the lymph-flow and therewith the resistance of the tissues to bacterial toxins. It is indeed a definite indication for prompt paracentesis or Saemisch's section ; otherwise the condition will go from bad to worse.

If there is a mucocele the lacrymal sac should be excised as soon as possible (*vide* p. 656).

Mycotic Hypopyon Ulcer. A rare form of hypopyon ulcer due to a fungus, the aspergillus fumigatus, is occasionally met with. In it the slough is dry in appearance, and is surrounded by a yellow line of demarcation, which gradually deepens into a gutter. As the name implies, there is an hypopyon. *Treatment* is the same as for other hypopyon ulcers.

Ring Abscess. See p. 447.

Phlyctenular Keratitis. It has already been pointed out that phlyctens are commonly found seated upon the limbus.

They may also occur within the corneal margin. The fact must be emphasised that the disease is essentially conjunctival, and when the cornea is affected it is the conjunctival element of the cornea, viz., the epithelium and the superficial layers immediately underlying it, which suffers. Phlyctenular keratitis does not necessarily result in ulceration, so that in these cases it is incorrect to classify it as a purulent keratitis, but it is convenient to consider the corneal manifestations of the disease under this heading because the complications and their treatment are similar to those of corneal ulcers in general.

Corneal phlyctens are localised infiltrations of exactly the same nature as conjunctival phlyctens. They cause more pain and reflex blepharospasm (photophobia) than do the conjunctival ones, symptoms which are worse in the morning. They may become absorbed without destruction of the overlying epithelium: in this case they cause no permanent opacity. The tendency for the epithelium to be destroyed or rubbed off is very great, and the denuded surface easily becomes infected, usually by staphylococci (*vide* p. 168). In this manner a small superficial ulcer is formed.

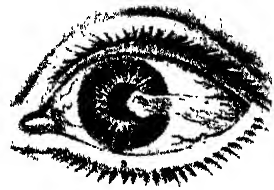


Fig. 133. — Fascicular ulcer, travelling inwards towards the centre of the cornea. (After Nettleship.)

The corneal phlycten is a grey nodule, slightly raised above the surface. If the epithelium breaks down and an ulcer is formed the surface becomes covered with polymorphonuclear leucocytes and looks yellow. The subsequent course depends probably upon the nature of the infection and the condition of nutrition of the patient. It may deepen rapidly and even perforate, though this is comparatively uncommon.

A very characteristic form of phlyctenular ulcer is the *fascicular ulcer* (Figs. 72 B, 133). This is a serpiginous ulcer which steadily creeps over the cornea, usually towards the centre, advancing slowly for weeks. It is supplied by a leash of vessels which lie in a shallow gutter and follow the advance of the ulcer. The ulcer starts near the limbus and heals on the peripheral side, while the central margin remains grey and infiltrated. As long as this infiltrated crescent is seen the ulcer is progressing. It always remains superficial and never perforates. When healing finally takes place the vessels gradually disappear, but the whole of the track of the ulcer remains as

are quite superficial, show little infiltration and no vascularisation; they form shallow round pits or facets about 2 mm. in diameter. There is little or no reaction, either in the form of lacrymation or photophobia. They do not spread either superficially or in depth, nor do they show any tendency to heal. When they finally heal they often leave clear facets, which only very gradually disappear. They appear to have nothing in common with phlyctenular keratitis except that both conditions are indications of defective nutrition. They are not uncommonly associated with trachoma.

They sometimes occur outside the centre of the cornea, and may perforate, allowing a knuckle of iris to prolapse. This prolapse should not be cut off, owing to the relatively large gap in the cornea, and the defective powers of repair in the debilitated patient.

Treatment must be directed especially to improving the general nutrition. A few weeks in a convalescent home in the country will effect more than any local treatment. Atropine and boric lotion are used locally. Trachoma, if present, must be suitably treated.

Keratomalacia is a rare disease in England, affecting badly nourished children, usually early in the first year of life. The conjunctiva becomes dry and shows xerotic spots (*vide* p. 193). The cornea becomes dull and insensitive; the haze increases and yellow infiltrates form. Finally the whole cornea necroses and may seem to melt away within a few hours. A characteristic feature is the absence of inflammatory reaction. In the rare cases in which the children are old enough to exhibit this symptom the disease commences with night blindness; they are able to see much better in the daytime than in the dusk. The children are usually extremely ill and very frequently die. Owing to their apathetic condition they do not close the lids, so that the cornea is continually exposed. Both eyes usually become affected. Streptococci have been found in the cornea and sometimes in the blood; in other cases the pneumococcus is present. Many of the children are syphilitic. Experiments on animals tend to show that the disease is due to the absence of fat-soluble vitamin A in the diet.

Treatment must be directed to the general health and environment. Cod-liver oil should be given or rubbed into the skin. Halibut-liver oil, 10 to 20 drops a day, or carotene in oil, 40 to 60 drops a day, are efficient substitutes for cod-liver oil. Subcutaneous injections of large quantities of normal saline solution are beneficial. The lids should be kept closed

under moist warm compresses. The nutrition of the cornea is sometimes benefited by the use of eserine.

Atheromatous Ulcers occur in old dense leucomata, especially such as have undergone degenerative changes resulting in the formation of hyaline fibrous tissue and calcareous deposits. Such scars have little vitality, and the deposits act as foreign bodies. They readily succumb to infection, the epithelium being badly nourished. When ulceration once begins it proceeds rapidly and deeply, with little or no effort at repair. Perforation takes place, and is often followed by panophthalmitis.

Treatment. The eye is frequently blind and disfiguring. In such cases it is well to excise it at once, thus relieving the patient of much unnecessary misery. If it is worth saving the condition must be treated on general principles.

Keratitis e Lagophthalmo occurs in eyes insufficiently covered by the lids. The epithelium of the exposed cornea becomes desiccated and the substantia propria hazy. Owing to the drying the epithelium is cast off, and the cornea falls a prey to infective organisms.

The condition is due to any cause which may produce lagophthalmia, *e.g.*, extreme proptosis as in exophthalmic goître or orbital tumour, paralysis of the orbicularis, and so on. The absence of reflex blinking is an important factor, as well as defective closure of the lids during sleep. Patients extremely ill from any disease are liable to this form of keratitis.

Treatment consists in keeping the cornea well covered. In mild cases it is sufficient to bandage the eyes at night. If possible the cause of the exposure must be removed. In the meantime it may be necessary to keep the lids closed with plaster and a bandage, or partially to sew them together (*vide* p. 227).

Chronic Serpiginous Ulcer (*Syn.—Rodent Ulcer, Mooren's Ulcer*). This is a rare superficial marginal ulcer, usually occurring in elderly people, and spreading, if not checked, over the whole cornea. It is accompanied by severe pain and lacrymation. It commences by one or more grey infiltrates. These break down, forming small ulcers, which spread and sooner or later coalesce. The ulcer undermines the epithelium and superficial lamellæ at the advancing border, forming a whitish overhanging edge, which is very characteristic. The base becomes quickly vascularised. It never perforates, but goes on with intermissions for months, until eventually a thin nebula is formed over the whole cornea, and sight is much diminished. There is

sometimes iritis, and very rarely a small hypopyon. In about a quarter of the cases both corneæ are affected, but not always simultaneously. The cause is unknown.

Treatment. The overhanging edge should be cut off with scissors, and then the whole surface of the ulcer, and especially the margin, should be well cauterised with the actual cautery, or with trichloracetic acid (10—20 per cent.), and covered by a conjunctival flap. The tension should be kept low by repeated paracentesis. A few cases have responded to repeated applications of absolute alcohol to the ulcers; to zinc ionization followed by covering with a conjunctival flap; or to β-rays (*vide* p. 187). More commonly treatment fails to stop the process, which has even been known to recur in the cicatrised cornea.

Neuroparalytic Keratitis occurs in some cases in which the fifth nerve is paralysed. It is relatively rare in nuclear and fascicular lesions within the central nervous system, unless the facial nerve is simultaneously involved. Nor does it occur in all cases of peripheral lesion; thus, if the Gasserian ganglion is removed or the fifth nerve injected with alcohol for trigeminal neuralgia with proper precautions only a small proportion of the cases get neuroparalytic keratitis. The disease has been known since the time of Majendie, and was attributed to special trophic impulses conducted by the nerve. This theory was combated, and the view advanced that the condition depends upon the loss of sensation in the eye. As a result, reflex blinking is more or less abolished, minute foreign bodies are not felt and therefore not removed, abrasions are unnoticed and untended, so that ulceration is readily induced and pathogenic organisms have free play. Neither of these theories satisfactorily accounts for the facts. It will be seen that the disease of the cornea usually has a very characteristic nature, quite different from the ulceration of neglected injuries. It is probable that the disease is due to irritative changes in or about the degenerating nerve, and that mere section or paralysis of the nerve is unable to produce the disease in the absence of such irritative conditions. Some light has been thrown on the "trophic" function of sensory nerves by the work of Krogh, Dale, Lewis and others (*vide* p. 17). They have shown that antidromic impulses and axon reflexes play a large part in the automatic control of the metabolism of the tissues supplied by these nerves. It is possible that excessive output of histamine-like substances may account for "trophic" skin and other lesions, such as neuroparalytic keratitis. On the other hand, the importance of reflex closure of the lids is

shown by the fact that keratitis occurs in all cases of paralysis of both fifth nerves. When only one is affected the consensual reflex from the other eye affords protection. Moreover the disease is more likely to occur if there is proptosis or paralysis of the orbicularis palpebrarum.

Besides these cases which result from radical treatment of trigeminal neuralgia, neuroparalytic keratitis is caused by intracranial tumours, gummatous basal meningitis, and fractures of the skull. (*See* Chap. XXIX.)

The characteristic feature of neuroparalytic keratitis is the desquamation of the corneal epithelium. The surface of the cornea becomes dull, and the epithelium is thrown off, first

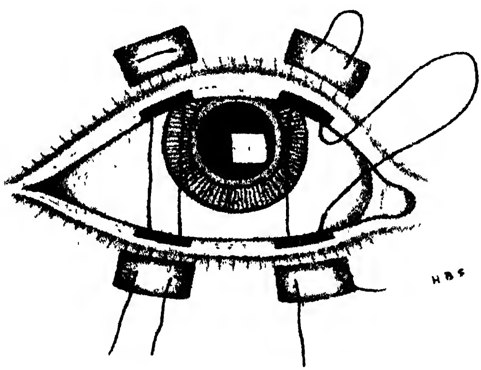


FIG. 134.—Tarsorrhaphy.

at the centre, then over the whole surface except a narrow rim at the periphery. The whole of the epithelium may peel off intact. The substantia propria then becomes cloudy and finally yellow, breaking down into a large ulcer, which is usually accompanied by hypopyon. There is no pain, owing to the anæsthesia, but ciliary injection is marked. A large perforation occurs if the case is not speedily treated. In any case the resulting leucoma is generally so large as to destroy useful vision. Relapses are the rule, the healed scar quickly breaking down again, and the whole process being repeated.

After complete removal of the Gasserian ganglion sensation is never regained.

Treatment. The ordinary treatment of corneal ulcer should be tried as a preliminary, special care being devoted to the

protection of the eye with a bandage. Improvement is often marked, but directly the bandage is relinquished the ulceration starts anew. In these cases it is best to suture the lids together, and to keep them sutured for a long period, only separating them when the conditions of the weather—warmth, absence of high winds, and so on—are favourable.

No anæsthetic is necessary, since sensation is lost in the conjunctiva and lids. The mucous membrane is dissected up from the margin of the lower lid just posterior to the lashes over rectangular areas about 6 mm. long on each side of the middle of the lid. The edge of the upper lid is similarly treated at the corresponding positions. Two mattress sutures are then passed through rubber sheet and the skin so that they come out at the posterior edge of the bare surface (Fig. 134)—not on the posterior surface of the lid where they would rub against the cornea. After being similarly carried through the skin of the other lid and rubber they are tied, the freshened surfaces being brought into contact. In a few days, if the stitches have not torn out, the lids will be firmly adherent.

The beneficial effect of suturing the lids on neuroparalytic keratitis is very striking. If, as often, bandaging fails, tarsorrhaphy will invariably succeed in stopping the process. Even if an hypopyon ulcer is present cicatrisation rapidly takes place and the hypopyon disappears. Recurrence of the keratitis, however, very frequently happens if the lids are separated, even after many months.

In the worst cases the eye is useless, and tends to become a source of perpetual trouble. It is then best to excise it, which may be done without any anæsthetic if the ophthalmic branch of the trigeminal nerve is completely paralysed.

Non-suppurative Keratitis. This occurs in two types, superficial and deep. The superficial forms include some purely symptomatic conditions, such as pannus. Some of them, such as herpes, may lead to the formation of ulcers and pass into the purulent type. The deep forms never suppurate. A considerable number have been shown and more have been suspected to be virus infections, the commonest of which are herpes febrilis, herpes zoster, superficial punctate keratitis in all its variants and disciform keratitis.

Herpes Corneæ. Herpes of the cornea occurs most commonly in herpes febrilis, more rarely in herpes ophthalmicus.

Herpes Febrilis. In herpes febrilis vesicles form upon the cornea quite similar in nature to those which may be present, but are not always so, upon the lips or angle of the nose. The patients

are usually suffering from some febrile condition, *e.g.*, influenza, pneumonia, whooping cough, &c., but it may be so trivial as to escape observation. It sometimes occurs after typhoid inoculations. The vesicles are scarcely the size of a pin's head and are often arranged in rows or groups. They quickly rupture, forming abrasions which heal rapidly, leaving no opacity. Usually, however, fresh crops of vesicles appear, and the condition may prove very obstinate. In severe cases, ulcers are formed which may be of dendritic type, or may be due to secondary infection. The acute stage is accompanied by great irritation, lacrymation, and blepharospasm. The ocular affection is usually unilateral and on the same side of the face as vesicles on the lips, &c. The cornea is generally not anæsthetic except at the spots attacked.

Herpes corneæ may be mistaken for phlyctenular keratitis. The former occurs usually in adults, the latter in children. The clear vesicles differ in appearance from the grey infiltrations of phlyctenular keratitis. After the vesicles have burst, the shape and the total absence of vascularisation are distinguishing features. They are then liable to be mistaken for traumatic abrasions, from which they are distinguished by their grouping, the crenated edges when several have coalesced, persisting shreds of the ruptured vesicles, and the absence of history of any injury.

FIG. 135. — Dendritic ulcer, somewhat diagrammatic.

The commonest form of herpes corneæ is the *Dendritic Ulcer*. In it the vesicular stage is rarely seen, the epithelial wall of the vesicle being quickly broken. Minute shallow clear facets, like abrasions, are found in the first stage. They may be easily overlooked. They generally cause much pain, lacrymation and blepharospasm. They may spread in all directions, coalescing with others and forming a large shallow ulcer with crenated edges. More often grey striæ extend in one or more directions, increase in length, and send out lateral branches, which are generally knobbed at the ends (Fig. 135). In this manner a dendritic figure, not unlike a liverwort, is formed. The surface over the infiltrates breaks down and an extremely irritating and chronic type of ulcer is produced, persisting with exacerbations for weeks or months. Generally only one or two of the infiltrates stains with fluorescein at any given time,

but fresh spots are continually being formed. It is often associated with frontal neuralgia. Such an ulcer may persist in spite of treatment for weeks or months, sending out fresh branches but never extending in depth. The disease not infrequently recurs.

Dendritic ulcer is really a manifestation of herpes febrilis. It sometimes occurs after prolonged treatment with arsenic, and also in subjects of malaria.

Treatment. In herpes corneæ the eyes should be protected with a bandage. When the vesicles have ruptured, atropine and warm compresses give most relief. It is sometimes necessary to use pantocain when the pain is acute. Emollient applications, parolein or sterile vaseline are sometimes grateful. Prolonged ulceration must be treated according to the type of ulcer, whether dendritic or septic. The general health must be attended to; quinine, valerian, salicylic preparations, *e.g.*, salicylates, aspirin, &c., are useful.

Dendritic ulcer may be carefully cauterised with iodine (7 per cent. iodine and 5 per cent. potassium iodide in alcoholic solution) on a swab, followed by instillation of pantocain, but pure carbolic acid is more efficacious. Absolute alcohol has been recommended for the purpose in these cases, but causes much pain after the pantocain has ceased to act. If caustics fail to stop the progress the actual cautery should be used. Atropine, &c., and a pad and bandage are used, but in many cases it is advisable simply to use smoked glasses and get the patient out in the fresh air as much as possible. Sometimes for no known reason eserine is successful when atropine has failed to produce a good result. The general health must not be neglected, especially as the patient often becomes very depressed.

Ultra violet light treatment has proved efficacious in some cases; the reaction is increased by previous instillation of 1 per cent. fluorescein.

Good results have been obtained by the administration of vitamin C (ascorbic acid). It should be given intravenously on alternate days from "Roche" ampoules containing 500 milligrams. Generally 4 to 6 injections suffice, but should be followed by 2 tablets of 250 milligrams by the mouth three times a day.

Herpes Zoster. In herpes zoster ophthalmicus one or more of the branches of the ophthalmic division of the fifth nerve (Fig. 136) is marked out by rows of vesicles or the scars left by them, exactly as in herpes zoster in

other parts of the body. The supra-orbital, supra- and infra-trochlear branches are nearly always involved;

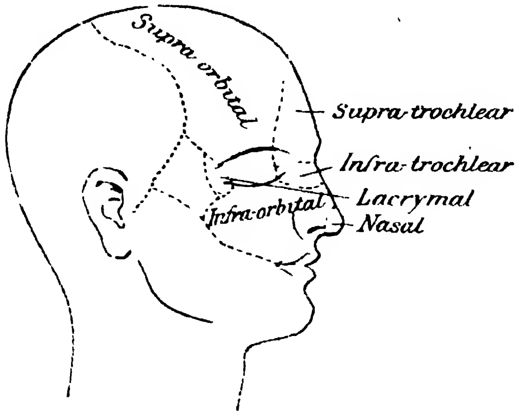


FIG. 136.—Distribution of the branches of the ophthalmic division of the fifth nerve on the face.

FIG. 137.—Herpes ophthalmicus.

frequently the nasal branch; only rarely the infra-orbital branch. It is very rarely bilateral. There may be fever and

malaise at the onset, and the eruption is preceded by severe neuralgic pains along the course of the nerves. These are so characteristic that they should arouse suspicion of the nature of the disease before the vesicles appear. The pain sometimes ceases after the outbreak of the eruption, but not always, and it may continue for weeks or months. The skin of the lids and other areas affected becomes very red and œdematous, so that the disease is often mistaken for erysipelas. The characteristic distribution and especially the strict limitation to one side of the middle line of the head should obviate this error. The vesicles often suppurate, bleed and cause small, permanent pitted scars. The active eruptive stage lasts about three weeks and is followed by some anæsthesia of the skin. Ocular complications arise during the subsidence of the eruption, but may be overlooked during the acute stage owing to the difficulty in examining the eye.

With the slit-lamp rounded spots, composed of minute white dots, which fuse into irregular areas, are seen. Vesicles are rare, but when they occur are exactly like those of the febrile form, and behave in the same manner. More commonly there is a diffuse deep infiltration of the cornea (*keratitis profunda*), associated with iridocyclitis (*q.v.*). The cornea is usually insensitive. This is tested by touching it with a wisp of cotton wool, and comparing with the opposite eye. The slightest touch is followed by reflex closure of the lids if the cornea is sensitive. The intraocular tension is not infrequently somewhat diminished in the early stage. The eye lesions are very obdurate and often persist long after the disease has otherwise passed away. In some cases there is associated paralysis of motor cranial nerves, especially the third, sixth and seventh. It usually passes off within six weeks. Facial palsy adds seriously to the risk of the eye owing to exposure.

Apart from permanent scarring of the cornea and the evil effects of iridocyclitis, anæsthesia of the cornea may persist for months. Nodules of scleritis may occur about two months after the disappearance of the rash (Doggart), and patches of atrophy in the iris are common. Quite acute pain, with impairment of sensitivity, may persist in the affected skin for months or even years.

Herpes ophthalmicus occurs at any age, but generally in elderly people. Not infrequently there is a history of contact with patients suffering from chicken-pox, and evidence is accumulating that the relationship is more than a mere coin-

cidence. It may account for the fact that herpes zoster is apt to occur in epidemics, which are more common in the spring and autumn than at other times.

The disease is due to lesions in the Gasserian ganglion of the same type as those found in the posterior root ganglia in herpes zoster, *i.e.*, microscopic thromboses and hæmorrhages similar to those found in the anterior cornual nuclei in anterior poliomyelitis. This disease shows many analogies on the motor side to herpes zoster and there is some evidence that posterior poliomyelitis is associated with the posterior root lesions of herpes (Lhermitte). Probably both are caused by infection, the lesions being due to a virus. Herpes ophthalmicus has been known to follow involvement of the Gasserian ganglion in a malignant growth, gummatous meningitis, or arsenic poisoning *e.g.*, with salvarsan. The cases associated with motor palsies, usually the 3rd nerve, and rare cases with optic atrophy, are also probably cases of symptomatic herpes and not due to the herpes zoster virus. In all cases the skin lesion is probably due to antidromic impulses liberating histamine-like bodies, and the spread of the vaso-dilatation may well be due to axon-reflexes.

Treatment. The eye should be carefully examined in every case of herpes ophthalmicus, the lids being separated by retractors if necessary. Oily drops or parolein and atropine should be instilled. Keratitis and iridocyclitis must be treated in the usual manner. The skin may be treated with cocaine ointment (1 per cent.) and dusted with starch powder. Quinine is generally given internally and aspirin to relieve the pain. If the pain is severe it may be relieved by intramuscular injection of pituitrin. The patient should be kept in bed during the acute stage.

Superficial Punctate Keratitis is an uncommon condition allied to herpes febrilis, and, like it, usually associated with influenza or catarrh of the respiratory tract; but no vesicles are formed. It commences as an acute conjunctivitis. At this stage, or after it has passed off, groups or rows of superficial, slightly raised, grey dots are seen scattered over the cornea, least at the periphery. They may or may not stain faintly with fluorescein. They may remain unchanged for months, but gradually disappear: some cases, however, pass on to disciform keratitis (*q.v.*). The disease affects one or rarely both eyes usually in young people, and is accompanied by irritation and lacrymation in the early stages, with some defect of vision in the later. Unlike recurrent erosion of the

cornea (*q.v.*), which may occur in similar circumstances without known trauma, it does not recur.

Corneal lesions of this type, caused almost certainly by a virus or different but related viruses, have the habit of recurring in mild or severe epidemics. The disease may run a protracted course, and it is only recently that the relation between the various outbreaks separated in time and space have been recognised. Epidemics have been common in India but rare in Britain; the most recent (*epidemic keratoconjunctivitis*), conspicuously associated with parotid adenopathy, had a widespread distribution between 1938 and 1944 in the United States and the Far East. In this epidemic various strains of virus have been isolated and cultured.

Treatment, apart from bland lotions and atropine, has so far been ineffective.

Keratitis disciformis, originally considered a milder inflammation of the same type as hypopyon ulcer, since it is occasionally accompanied by a small hypopyon, is probably, in most cases, the result of the extension of a virus infection into the substance of the cornea. It has long been known to result from vaccinia affecting the lid margins, or from herpes corneæ, and not infrequently develops from a superficial punctate or macular keratitis. It is characterised by a central grey disc lying in the middle layers of the substantia propria. The disc is sharply defined and often shows several concentric grey lines, rather like a target. In the centre there is usually a denser "bull's eye." The slit-lamp shows thickening of the cornea (Vogt), and often folds of Descemet's membrane. This form of keratitis is not common: it occurs generally in adults and is unilateral. It is accompanied by moderate irritation, which, however, persists for several weeks or months, leaving a permanent opacity. Owing to the central situation vision is considerably impaired. There is no ulceration, but the inflammation is probably caused by ectogenous infection through a defect in the epithelium. It is little amenable to treatment.

In **acne rosacea**, generally in elderly women, keratitis associated with much irritability and lacrymation may occur. In addition to slight muco-purulent conjunctivitis (*vide* p. 149), greyish white infiltrates and small ulcers appear in the cornea. They are very intractable and frequently recur. In severe cases there is also iritis. It is not due to ariboflavinosis (*v.* p. 249).

The local treatment should be similar to that for phlyctenular keratitis, and calomel insufflation gives good results; mild application (one-third of an erythema dose) of X-rays has been

found beneficial (Greeves); the essential treatment, however, is that of the skin condition, combined with suitable diet.

Interstitial Keratitis (*Syn.—Parenchymatous Keratitis*) is a deep keratitis usually affecting children between the ages of five and fifteen, the subjects of congenital syphilis. It is commoner in girls than boys.

After slight irritative symptoms, with some ciliary congestion, one or more hazy patches appear in the cornea, near the margin or towards the centre (Plate VII., Fig. 1). If they are near the margin they push forwards towards the centre; if at the centre, others appear and fuse, until finally the whole cornea looks lustreless and dull. Minute examination shows that the patches are in the deep layers and are made up of denser spots and fine streaks. In two to four weeks the whole cornea is hazy with a steamy surface, giving a general appearance like that of ground glass. Denser spots can always be seen in the general mist. In the severest cases the whole cornea becomes quite opaque, so that the iris is hidden; as a rule the iris can be seen dimly.

Meanwhile vascularisation has occurred. It is of deep type (*vide* Fig. 73, p. 91), consisting of radial bundles of brush-like vessels. The larger marginal plaques may be very vascular; as they are covered by a layer of hazy cornea, their bright scarlet is toned down to a dull reddish pink ("salmon patches"). The separate vessels can only be seen by magnification. The small salmon patches are often crescentic; when larger they are sector-shaped. The opacity extends a little beyond the vessels, which seem to push the opacity in front of them. In the acme of the condition the vessels run in radial bundles almost, but seldom quite, to the centre of the cornea. Vascularisation is probably brought about for the supply of protective substances which cannot reach the cornea by the normal process of diffusion. There is often a moderate degree of superficial vascularisation, greater in some cases than in others, but never extending far over the cornea. The conjunctiva may be heaped up like an epaulette at the limbus, so that some slight resemblance to phlyctenular keratitis may be seen. Indeed, it is probable that these patients are both syphilitic and tuberculous. This florid stage usually lasts for two to four months.

After the disease has reached its height it commences very slowly to subside. The cornea clears from the margin towards the centre, which may long remain hazy, though it too finally

chronic synovitis of the joints, especially the knee joints, may be present.

Evidence of acquired syphilis may be sought in the parents, *e.g.*, history of miscarriages, &c. In cases of doubt, the Wassermann test should be applied.

As already mentioned, interstitial keratitis also occurs in acquired syphilis, and it has been attributed to malaria, myxœdema, trypanosomiasis, &c. It frequently occurs in foxhounds and other highly-bred dogs, and I have seen it in dogs after the thyroid gland has been removed.

Treatment. It is usual to order antisyphilitic remedies, but, as in parasyphilitic diseases of the central nervous system, it is doubtful if they have any influence over the course of the disease, partly because the cornea is non-vascular. Mercury inunctions, or powders or pills of mercury combined with chalk and ipecacuanha or perchloride of mercury may be given. Iodide is best administered to children in the form of syrup of iodide of iron, and may be combined with syrup of phosphates as a tonic. Salvarsan and its substitutes have proved disappointing. The administration of thyroid gland has proved serviceable in some cases. Cod liver oil or maltine may be used when there is a tuberculous element about the condition, and in all cases general hygienic régime must be instituted.

Local treatment consists in guarding against the evil effect of the uveitis which is an invariable accompaniment of the disease. Atropine is ordered as a routine measure, with the double purpose of keeping the ciliary body and iris at rest and preventing the formation of posterior synechiæ. There is often great difficulty in getting the pupil to dilate, probably owing to defective penetration of the drug through the diseased and vascularised cornea. Hot bathings or radiant heat should be used frequently in the acute stage. In obstinate cases with much lacrymation and blepharospasm, especially if the pupils will not dilate with atropine, leeches applied to the temple do good (*vide* p. 269); or mydricain (*vide* p. 694) may be tried. Smoked glasses are ordered.

In some cases pain and blepharospasm are so severe that no relief is obtained by the usual measures. The retro-ocular injection of 1·5 c.c. of novocain (4 per cent.) into the region of the ciliary ganglion, followed seven minutes later by 1 c.c. of alcohol (40 per cent.) is effective. The pain and blepharospasm are relieved, vascular congestion much reduced, and the child is able to tolerate light. In some cases symptoms recur after two weeks, and a testimony as to

the efficacy of the treatment is that the child may ask for another injection. No ill effects have hitherto occurred.

In later stages the means used for clearing corneal cicatrices (*vide* p. 207), depending essentially upon improving the lymph flow through the cornea, are also indicated for the opacities of interstitial keratitis. Prolonged use of yellow oxide of mercury ointment, combined with atropine in the earlier stages, is commonly made: it should be well massaged into the eye.

Other forms of deep keratitis, *e.g.*, the central deep keratitis of adults, senile marginal deep keratitis, sclerosing keratitis (*vide* p. 253) occur.

Keratitis profunda is the name given to a central deep infiltration of the cornea of indefinite origin. Such a condition occurs

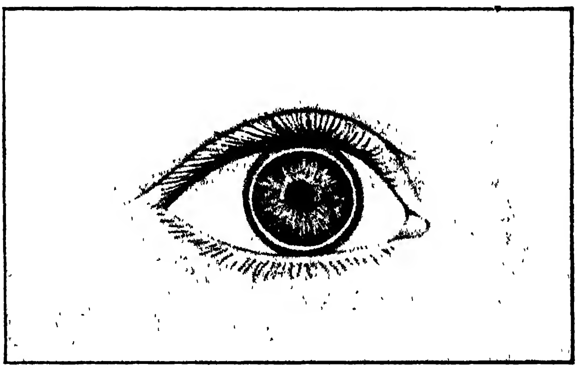


FIG. 145.—Arcus senilis.

after contusion of the eye, and in this case usually clears up rapidly. In other cases a deep grey opacity, seen on magnification with the loupe to be composed of dots and striæ, occurs in the centre of the cornea in adults. There is little irritation, and very little, if any, vascularisation. It begins to clear up after four to eight weeks, but may leave permanent diffuse opacities. It has been attributed to cold, malaria, and other causes. It should be treated with the same local remedies as for interstitial keratitis.

Keratitis marginalis profunda occurs rarely in old people, commencing as a greyish yellow infiltration, one or two millimetres broad, continuous with the sclera. It is usually limited to the upper part of the cornea, but occasionally forms a complete ring. The slight irritation subsides in a week or two, leaving a permanent opacity, resembling arcus senilis, but unlike it, continuous with the sclerotic.

Degenerative Changes in the Cornea

Arcus senilis is a lipoid degeneration of the cornea met with in old people (Fig. 145). It commences as a crescentic grey line concentric with the upper and lower margin of the cornea. The extremities of the crescents finally meet, and an opaque line, thicker above and below, is formed completely round the cornea. It is characterised by being separated from the margin by a narrow line of comparatively clear cornea. It is sharply defined on the peripheral side, fading off on the central. It is never more than about 1 mm. broad, and is of no importance, either from the point of view of vision or of the vitality of the cornea.

Arcus juvenilis is exactly similar to arcus senilis, but is a rare condition found in children. Even arcus senilis may develop at a comparatively early age, but the juvenile condition is probably congenital. It is of no importance. The characteristic diagnostic feature of both these opacities is the presence of a line of clear cornea between them and the limbus. This is occasionally found in old sclerosing keratitis, but in this case the opacity is usually localised to some one part of the cornea and extends farther towards the centre.

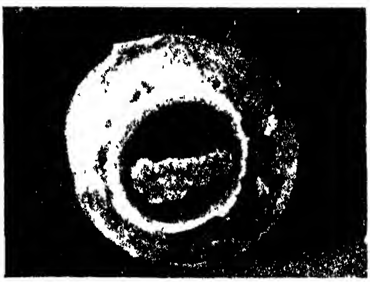


Fig. 146.—Band-shaped opacity of the cornea, from an eye with iridocyclitis.

Band-shaped Opacity (*Syn.—Transverse Calcareous Film, Zonular Opacity*). This is a common condition in old, blind, shrunken eyes. It is due to defective nutrition and exposure. It lies entirely in the interpalpebral area, commencing at the inner and outer sides, and progressing until it forms a continuous band across the cornea (Fig. 146). Near the corneal loops, just inside the limbus, the cornea is generally relatively clear, as in so many degenerative conditions—probably owing to the better nutrition close to the blood-vessels. The condition is due to hyaline degeneration of the superficial parts of the substantia propria, followed by the deposition of calcareous salts.

As a rare condition it is found in otherwise healthy eyes, sometimes as a horizontally oval area in the palpebral fissure, usually in both eyes.

Treatment. In the rare form last mentioned, improvement of vision may be obtained by scraping off the opacity, which is usually calcareous and quite superficial. In the common form

the eye is blind, and nothing remains but to remove it if it is painful or unsightly.

Other Degenerative Changes are frequently met with in old leucomata, anterior staphylomata, and so on. They consist of hyaline degeneration, calcification, &c. Such scars are liable to a serious form of ulceration (*vide* p. 225).

Rare degenerative conditions are *nodular* and *reticular* (grill or lattice-like) *opacities*. They occur as a familial disease, usually picking out the young males of a family and commencing at about the age of ten or twelve. Opaque dots or forked lines appear under the epithelium in the centre of the cornea and slowly increase in number, but never invade the ring of cornea close to the margin. There is little inflammatory reaction and the cause is unknown. Vision is gradually obscured and treatment is of little avail. I have seen grey mosslike opacities, apparently of similar nature, with the same distribution in the corneæ of elderly people. Examination with the slit-lamp shows that some of these cases have thickened corneal nerves and nodular swellings of the nerve-endings. Others show folds or ruptures of Bowman's membrane.

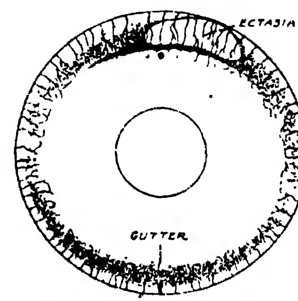


FIG. 147.—Senile marginal atrophy (Koby).

Senile marginal atrophy, in which a gutter forms very slowly in the periphery of the cornea in the situation of an arcus senilis, occurs rarely in one or both eyes of old people. The gutter may become ectatic (Fig. 147).

ECTATIC CONDITIONS

It has already been stated that ectatic conditions of the cornea may result from inflammation, viz., keratectasia (*vide* p. 201), and anterior staphyloma (*vide* p. 203). Two forms of ectasia of non-inflammatory origin are known, viz., keratoconus and keratoglobus.

Anterior Staphyloma is a protuberant cicatrix arising from a prolapse of the iris; it may be partial or total (p. 203). It may follow a perforating wound, but is usually due to perforation of an ulcer, especially such as is caused by ophthalmia neonatorum. The primary protrusion occurs at the moment of prolapse. Cicatrisation follows, and in the case of small prolapse may lead to flattening of the scar. In other cases of small, and in all cases of large, prolapse the con-

traction of the scar tissue is insufficient to bring this about, and the soft cicatrix yields to the normal intraocular pressure. Generally the prolapse of the iris leads to blocking of the angle of the anterior chamber, the intraocular tension rises, and the cicatrix yields still more, or if it was previously flat, secondary protrusion takes place.

Partial staphylomata are usually conical, rarely hemispherical; they usually extend to the margin on one side. Total staphylomata are usually hemispherical, rarely conical. There is invariably a rim of cornea around the pseudo-cornea, this rim being well nourished by the peripheral blood vessels and never necrosing through ulceration. The thickness of the staphyloma varies very greatly in different cases, and often in different parts of the same staphyloma. In the latter case bands of cicatricial tissue develop, while the intermediate parts project; in this manner a racemose staphyloma is produced. Owing to the rise of intraocular tension the whole eyeball expands, especially in children, in whom the walls are relatively plastic. If the lens has not been expelled when the ulcer perforated, as sometimes happens, it is flattened, the expansion of the ciliary ring causing stretching of the suspensory ligament. Owing to contact of the lens with the inflamed cornea after perforation the anterior capsular cells of the lens often proliferate and form an anterior capsular cataract (*q.v.*). The high intraocular pressure also causes cupping of the optic disc.

The pseudo-cornea is formed by organisation of the exudates on the surface of the prolapsed iris. It consists of fibrous tissue covered by epithelium, and lined by rarefied iris pigment epithelium. The epithelium on the anterior surface is often very thick and epidermoid; the fibrous tissue often undergoes degeneration. The anterior chamber is obliterated in total staphyloma, while the posterior chamber is enormously enlarged, and filled with yellow albuminous fluid.

The sight is always diminished, and in total staphyloma is reduced to perception of light or total blindness. The eye may project between the lids, so that a xerotic condition is set up and atheromatous ulcers may form. Ectropion of the lids may occur mechanically. The tension is raised, either as the cause of the protrusion, or as a result of the blockage of the angle of the anterior chamber (Chap. XIV.). This may lead to pain. The staphyloma may be so thin that rupture occurs on the least injury, and may be several times repeated.

Treatment. Total anterior staphyloma is best treated by excision of the eye, with or without the insertion of a glass

globe in Tenon's capsule. Patients will often prefer to keep the eye if it is painless and not too disfiguring.

Various methods of ablation, in which the anterior part of the eye is removed and the contents scooped out, have been devised as an alternative for excision. They give a movable stump on which to set an artificial eye, but they are open to the objection that they are not entirely free from the danger of causing sympathetic ophthalmia (*q.v.*).

Treatment of partial staphyloma is directed to obtaining flattening of the cicatrix, preventing or relieving increased intraocular tension, and improving sight. The attempts which should be made at the early stage have already been described (*vide* p. 214). Iridectomy is sometimes advisable with a view to improving vision and preventing or curing glaucoma (*q.v.*). It cannot be performed at the site of the staphyloma, since there is no anterior chamber here, but must be done at the clearest part of the cornea.

Keratectasia differs from anterior staphyloma in that the iris takes no part in it. Its causation and prevention have already been discussed (*vide* p. 201). Sometimes the whole cornea expands, producing a condition which is almost identical with the keratoglobus (*vide* p. 246) of infantile glaucoma, though due to a different cause. When it is fully developed treatment is useless.

Keratoconus (*Syn.—Conical Cornea*) is probably due to a congenital weakness of the cornea, though often it only manifests itself after puberty, generally in girls. The cornea is thin and weak near the centre, and gradually bulges forwards more and more; the apex is always slightly below the centre. Sometimes it pulsates synchronously with the arterial pulse, and this may cause a subjective apparent pulsation of the objects looked at. The pulsation may be demonstrated with Schiötz' tonometer. The cornea is at first perfectly transparent, and vision is impaired through the protrusion and alteration in curvature. If advanced the conical shape is easily recognised in profile. In the less advanced cases distortion and diminution in the size of the corneal reflex over the centre is the chief guide. These changes are best seen when the reflex from a large flat disc painted with broad concentric black and white lines (Placido's disc or keratoscope) is observed through a hole in the centre of the disc, or when the cornea is examined with the ophthalmometer. With the ophthalmoscope mirror at a distance of 1 metre a ring of shadow concentric with the margin is seen on the red reflex, altering its position on moving the mirror.

It is due to a zone through which few rays pass into the observer's eye owing to the emergent rays on the central side being convergent whilst those on the peripheral side are divergent.

The patient becomes myopic, but the error of refraction cannot be satisfactorily corrected with ordinary glasses owing to the hyperbolic nature of the curvature. The condition is almost invariably bilateral, though frequently more advanced on one side than the other. It may be slight and very slowly progressive, or the reverse. In the later stages the apex shows fine more or less parallel striæ, anastomosing at acute angles, best seen with the slit-lamp; and also discrete opacities which become confluent. A brownish ring, probably due to hæmosiderin, may form in the epithelium encircling the cone (Fleischer's ring). Sometimes there are ruptures in Descemet's membrane. Ulceration, rupture of the cornea, increase of tension, and so on, do not occur.

Treatment. In the early stages every effort should be made to improve vision with glasses and the progress should be carefully watched. In those cases in which they could be borne contact glasses (*vide* p. 538) have been very beneficial. Various methods have been adopted to stop the process. Miotics are probably useless. The best treatment is cauterisation of the apex with the actual cautery. The cauterisation at the extreme apex must be deep, and perforation here has been advocated. The latter procedure is not without danger to the eye from the formation of a corneal fistula, anterior synechia, infection, &c. The scarring from cauterisation is much less than might be anticipated, but it may be advisable to do an optical iridectomy if vision is still very bad.

Keratoglobus (*Syn.—Megalocornea*) is a hemispherical protrusion of the whole cornea, occurring bilaterally in males: it is familial and hereditary. It differs from buphthalmia (*q.v.*) in that the intraocular pressure is normal, the cornea clear, the angle of the anterior chamber normal, and there is no cupping of the disc. It appears to be a congenital overgrowth, and is not infrequently associated with arachnodactyly.

Symptomatic Conditions

There are many pathological conditions of the cornea which are merely evidence of disease in other parts of the eye or of extension of disease. Some are often described as true diseases of the cornea, notably as forms of "keratitis." This

involves a wrong principle and a misuse of terms which can only lead to confusion. Since it is of great importance to distinguish these conditions from primary affections of the cornea, both from the points of view of diagnosis and treatment, it will be well to review the more common here.

In **glaucoma** there is nearly always uniform diffuse bluish haze of the whole cornea. It is due to alterations in the refractive conditions of the corneal elements, brought about by the increased intraocular pressure, and not to any gross pathological change. If the condition persists the cornea becomes hazy throughout, and this haziness, unlike the former, does

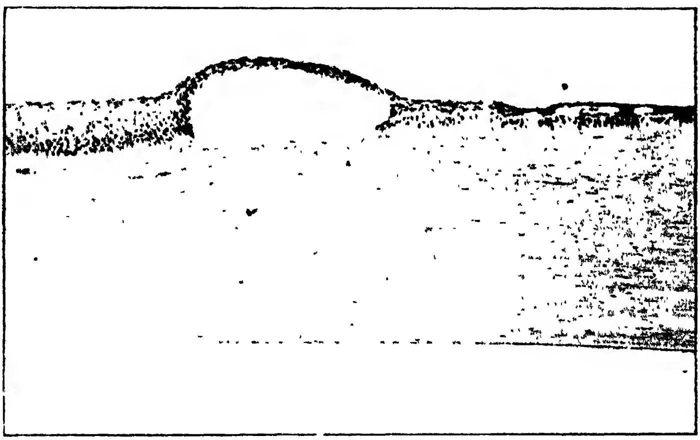


FIG. 148.—Œdema of the cornea and vesicular "keratitis," from a glaucomatous eye (× 60).

not immediately disappear when the intraocular tension is relieved. Here there is definite œdema, due to the impediment which prolonged tension causes to the diffusion of lymph. It manifests itself first in the epithelium, which becomes steamy and stippled. Sections show that this is due to accumulation of fluid in and between the cells, especially the basal cells (Fig. 148). Fluid also accumulates between the lamellæ and around the nerve fibres. If the œdema lasts for a very long period, as in eyes blinded with absolute glaucoma, the epithelium may be raised into vesicles or bullæ (*vesicular or bullous "keratitis"*). The evidence of prolonged tension makes it easy as a rule to distinguish this condition from

herpetic vesicular keratitis; moreover the vesicles or bullæ have firmer epithelial walls and show less tendency to burst.

Nearly allied to bullous "keratitis" is the formation of epithelial threads, which adhere to the cornea by one end, while the other, which is often club-shaped, hangs down free. This is commonly called *filamentary* "*keratitis*." It occurs in the same conditions, *i.e.*, usually associated with glaucoma, but it may be seen rarely without any apparent cause in cases of the herpetic type.

Keratic precipitates, usually badly termed "keratitis punctata" or "k.p." in England, are often deposited upon the back of the cornea in cyclitis and iridocyclitis. The greatest care must be taken not to overlook them, since they may be almost the only objective sign of serious disease. They may be on the back of a clear cornea or the deeper layers may be infiltrated as a result of the iridocyclitis; thus, they are not uncommon in interstitial keratitis. Their appearance and nature will be described in discussing their cause (*vide* p. 273).

Opacities of the cornea are often secondary or symptomatic. Such are the grey or white, usually tongue-shaped marginal opacities which follow scleritis. Owing to their resemblance to the sclerotic they are called *sclerosing* "*keratitis*" (*vide* p. 253).

Congenital opacities of various kinds are sometimes met with. Many are not truly congenital, but are due to injury received at birth. Birth injury of the cornea takes the form of temporary diffuse opacity due to œdema, or of permanent vertical grey linear opacities due to ruptures of Descemet's membrane.

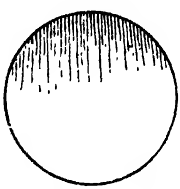


FIG. 149. — Striate opacity of the cornea, following section of the cornea above for extraction of cataract.

Striate opacity occurs in various forms. The commonest form is that seen after operations upon the globe in which a peripheral corneal section has been made, as in cataract extraction. Here, when the section is above, delicate grey lines run down vertically from the wound and may pass completely across the cornea (Fig. 149). They can be seen clearly only by magnification. They disappear spontaneously as the wound heals. They are due to slight folding of the cornea, whereby Descemet's

membrane and the adjacent lamellæ become wrinkled. Radial striæ are seen around wounds or ulcers; they are partly referable to the same cause, partly to distension of the interlamellar spaces by œdema. The fine hatching which is seen around ulcers and sometimes after tight bandaging is to be referred to similar causes.

Opacities of the cornea may be due to improper treatment. One of the commonest is due to *deposits of lead salts* upon an abrasion or ulcer. An insoluble, densely opaque white film is precipitated and adheres very firmly. The spot is sharply defined and looks like white paint. Probably the deposit is always thrown off eventually, but a very long time may elapse. An attempt should be made to scrape it off, but it is wisest never to use lead lotions at all in the treatment of any affections of the eye. They can always be replaced by equally efficient substitutes.

White Ring Opacities (Coats). Occasionally a ring or oval, about 0·5 mm. in diameter, composed of very dense white spots, occur on Bowman's membrane. The cause is unknown, they do not interfere with vision.

Pigmentation of the cornea may also occur from improper treatment. Prolonged use of silver nitrate as a paint or in the form of drops is followed by dark brown staining of the conjunctiva and slight staining of the cornea. The condition is called *argyrosis* (*vide* p. 193), and in the conjunctiva is found to be due to impregnation of the elastic fibres with metallic silver; in the cornea Descemet's membrane is stained. It is permanent; hence silver solutions, including protargol, argyrol, &c., should never be ordered as drops or for use at home, at any rate without stringent injunctions.

Blood in the cornea is rare. It may occur as a bright red spot quite superficially at the margin or as a greenish or rusty stain over the whole surface. In the latter case it is derived from blood in the anterior chamber, associated with high tension—a relatively infrequent combination (*vide* p. 438).

Ariboflavinosis. Deficiency of riboflavin (vitamin B_2 or G) in the diet leads to peripheral corneal vascularization, due to proliferation of capillaries from the normal sclero-corneal loops, and photophobia. The vessels are best seen with the slit-lamp (*v.* p. 98), but their diagnostic importance has been exaggerated. Ariboflavinosis should not be diagnosed in the absence of other signs of deficiency. In the fully developed condition the lips are purplish and cracked at the angles (*cheilosis*); the tongue is

inflamed (*glossitis*) and fissured, with enlarged papillæ. There is seborrhœa of the naso-labial folds, eyelids and ears ; and follicular keratosis of the forehead, malar eminences, and chin. Food rich in vitamin B_2 should be given (*vide* p. 698).

Tumours of the cornea, so-called, are probably always secondary extensions, most commonly from the conjunctiva, the limbus being a favourite situation for these growths (*vide* p. 194).

CHAPTER XII

Diseases of the Sclerotic

Redness of the white of the eye is caused by a variety of conditions. The commonest is conjunctivitis; the next common some inflammation of the anterior part of the uveal tract, viz., the iris and ciliary body. Some of the distinctive characteristics of the redness in conjunctival and ciliary inflammation have already been pointed out (*vide* p. 83). Redness of the white of the eye may also be caused by inflammation of the sclerotic, and it is a frequent error among beginners to ascribe the other forms to this disease. It must therefore be borne in mind that episcleritis and scleritis are relatively uncommon.

Inflammation of the Sclerotic

Two forms of inflammation of the sclerotic are described, superficial or episcleritis, and deep or scleritis. They might equally well be considered mild and severe forms of the same disease, but the distinction is convenient clinically since they usually differ in the course they take.

Episcleritis is an inflammatory affection of the deep subconjunctival connective tissues, including the superficial scleral lamellæ. A circumscribed nodule, which may be as large as a lentil, appears usually two or three millimetres from the limbus (Plate VII., Fig. 2). It is hard, immovable, and very tender, the conjunctiva moving freely over it. It is traversed by the deeper episcleral vessels, and therefore looks purple, not bright red. It is extremely chronic, never ulcerates, and may be entirely absorbed, but more frequently leaves a slate-coloured scar behind, to which the conjunctiva is adherent. The cornea and uveal tract rarely participate in the inflammation.

There may be little or no pain, but usually there is a feeling of discomfort and tenderness on pressure, and severe "neuralgia" is often complained of. The nodule becomes gradually absorbed in the course of days, or, more frequently, weeks,

but during the process of absorption, or soon after, fresh nodules of the same type arise. In this manner the disease may drag on for months. Both eyes are frequently affected. In the worst cases the disease extends into the deeper parts of the sclerotic, and thus passes almost imperceptibly into scleritis.

Anatomically dense lymphocytic infiltration of the subconjunctival and episcleral tissues is found.

Rheumatism and gout were commonly indicated as the chief causes of episcleritis, but it is now generally regarded as an allergic reaction to an endogenous toxin (tuberculous, streptococcal, etc.). A history of acute rheumatism is rarely obtained; more commonly there has been well-marked "muscular rheumatism," which is to be ascribed to septic absorption (*vide* p. 274). Tuberculous or syphilitic patients seem predisposed, and almost invariably there is some cause of general debility. It is commonest in women.

Transient but frequently repeated attacks of episcleritis sometimes occur, generally in gouty people (*episcleritis periodica fugax*).

Treatment. General treatment is of more avail than local, and should be directed in the first place to the elimination of focal infection and the desensitisation of the patient (*e.g.*, by tuberculin, a streptococcal vaccine, etc.). Even in cases in which no history of rheumatism can be elicited, salicylic preparations—salicylates, salicin, aspirin—seem to do good, and should be tried. If they fail resort should be had to iodides.

The most useful local treatment is massage by the finger applied to the upper lid. A simple boric ointment may be used or a weak yellow oxide of mercury ointment, but strong stimulants can rarely be borne. In the more severe cases warm compresses, dionin, and leeches to the temple should be employed. Every effort must be made to build up the constitution, and success often depends upon these measures.

Scleritis is rarer than episcleritis. There are usually nodules, or a single nodule, but the area affected is much less circumscribed. The swelling is at first dark red or bluish, later it becomes pale purple and semitransparent, like porcelain. It may extend entirely round the cornea, forming a very serious condition known as *annular scleritis*. Scleritis differs from episcleritis in that the cornea and uveal tract are involved, some iritis, but more cyclitis and anterior choroiditis being present. There is no ulceration, but much absorption, so

that the sclerotic is thinned, a dark purple cicatrix being formed, which is often too weak to withstand the intraocular pressure, so that ectasia follows (*ciliary staphyloma*). In many cases of diffuse deep scleritis hard whitish nodules develop in the inflamed zone. They are the size of a pin's head and lie beneath the conjunctiva, all at about the same distance from the corneal margin; they disappear without disintegrating.

Anatomically scleritis is the same as episcleritis, but extends deeper; there is dense lymphocytic infiltration of the sclera, the lamellæ being separated by cords of cells.

Both eyes are usually affected. Young adults are the most common subjects (cf. Episcleritis), and women more often than men. The causes are obscure, but of the same type as in episcleritis. It is often associated with disturbance of menstruation.

Scleritis is most serious on account of its sequelæ and complications. Uveitis of some kind is probably an invariable accompaniment. It is uncertain whether it may be a result or a cause of the scleritis; most probably it is neither, but both are due to a common cause. This cause is probably the absorption of toxins from some septic focus. It is often difficult or impossible to find the focus, but special attention should be directed to the nasal sinuses and generative organs, the latter especially in women. In other cases the alimentary canal is probably at fault, and many of the drugs which have been found beneficial probably act chiefly by disinfecting the intestinal tract. In some cases the disease is undoubtedly tuberculous and in others syphilitic. Stock has produced scleritis, sclerosing keratitis and lesions in the uveal tract by the intravenous injection of tubercle bacilli in rabbits. There is little doubt that tubercle plays a larger part in disease of these structures in man than has hitherto been recognised. Ciliary staphyloma leads to distortion of the globe, and vision is impaired by it and by the many deleterious effects of the uveitis. Secondary glaucoma often follows.

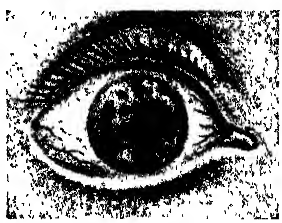


Fig. 150.—Sclerosing keratitis. (After Nettleship.)

Apart from these complications scleritis nearly always extends to the cornea, causing *sclerosing keratitis* (Fig. 150).

An opacity develops at the margin of the cornea near the scleritic nodule. It is approximately triangular or tongue-shaped, the rounded apex being towards the centre of the cornea. Similar opacities may develop farther from the margin and even at the centre. The opacities are grey or greyish yellow, becoming denser until they exactly resemble the sclerotic—hence the name. They are indeed due to changes in the substantia propria, which is embryologically a specially differentiated part of the sclerotic. There is little or no vascularisation, and ulceration never occurs. Some clearing from the centre towards the periphery, as well as near the marginal corneal loops (*vide* p. 212), occurs, but the densest parts usually persist as bluish clouds. The whole margin of the cornea may become opaque like the sclerotic, but the pupillary area almost invariably escapes.

Treatment is the same as for episcleritis. When tubercle is suspected injections of tuberculin may be employed. Uveal complications must be treated with atropine, &c. (*See* Chapter XIII.) Ultraviolet light sometimes has a good effect on sclerosing keratitis and good results have followed administration of vitamin C (*vide* p. 230).

Gumma of the Sclerotic is uncommon. It may be indistinguishable in appearance from scleritis, or it may take the form of nodules of various sizes, situated near the limbus, extending backwards to the equator, or even giving rise to an annular scleritis. Gumma may spread to the interior of the eye or a gumma of the ciliary body may spread outwards and involve the sclerotic. Unless active antisyphilitic treatment is adopted early and carried out thoroughly the eye is very likely to be lost from uveal complications, ciliary staphyloma, or phthisis bulbi.

The diagnosis depends upon the history, co-existing signs of syphilis, and the application of the Wassermann test.

Tubercle of the Sclerotic may take the form of a scleritis, may be an extension from the conjunctiva, iris, ciliary body, or choroid, or may be primary, forming a localised nodule which caseates and ulcerates. It should be excised or scraped and the tissue examined for tubercle bacilli.

Annular Scleritis, as already mentioned, may be a form of ordinary scleritis or of gummatous scleritis. A severer type is sometimes known as brawny scleritis and is characterised by a brownish-red, gelatinous-looking swelling surrounding the cornea and extending back towards the equator. It generally occurs in old people and is fortunately rare, for it is little

amenable to treatment. Some cases, but not all, give a positive Wassermann reaction.

Ulceration of the sclerotic is always secondary, either from without or from within. Extension from the conjunctiva is almost always due to tuberculous ulceration, rarely syphilitic. Extension from within is almost always from the iris or ciliary body and is usually tuberculous in the case of the iris, syphilitic in that of the ciliary body. Ulceration of malignant growths which have perforated the sclerotic also occurs—sarcoma of the iris or ciliary body, retino-blastoma. All these conditions are rare.

Blue Sclerotics. The sclerotic is bluish in babies, but a much more pronounced blue coloration is sometimes seen in several members of the same family as an hereditary condition, and persists throughout life. A curious and hitherto unexplained feature of these cases is that the patients in many of the families also suffer from fragilitas ossium and deafness. The sexes are about equally affected; only those affected can transmit the disease. In a case examined microscopically the sclerotic was about one-third the usual thickness; the cornea was also thin and Bowman's membrane was absent. The condition has been attributed to parathyroidism.

CHAPTER XIII

Diseases of the Iris and Ciliary Body

THE uncouth term *uveitis* has the merit of emphasising an important fact, viz., the close relationship which exists between the anatomically distinct parts of the uveal tract. It draws attention to the frequency with which inflammatory processes involve the tract as a whole, and are not strictly limited to a single part. This feature is particularly well exemplified in inflammation of the iris and ciliary body. Probably iritis never occurs without some cyclitis, nor, *a fortiori*, cyclitis without some iritis. The disease is called iritis or cyclitis according as the iris or ciliary body appears clinically to be the more affected. The same disposition is also seen with regard to the choroid, though in less degree. General uveitis is commonest in the more chronic types of inflammation, but it is probable that the ciliary body is often involved in many cases which we are accustomed to regard as pure choroiditis.

For convenience of description it is best to consider diseases of the various parts of the uveal tract separately, but the anatomical, physiological, and pathological continuity of the parts can be scarcely too forcibly insisted upon.

INFLAMMATION OF THE IRIS AND CILIARY BODY

Iritis. In order that iritis and the special dangers which attend it may be thoroughly understood, it is necessary to remember the anatomical arrangements of the iris and the pathological changes which occur in it. The iris is practically a diaphragm of blood vessels and unstriped muscle fibres held together by a very loose, spongy stroma. In its perpetual movements the pupillary margin slides to and fro upon the lens capsule. The more the pupil is constricted the more of the posterior surface of the iris is in contact with the lens capsule; when fully dilated the iris probably does not touch the lens at all.

Inflammation of the iris is fundamentally the same process as occurs in other connective tissues: it consists in dilatation

of the blood vessels, impairment of the capillary walls, exudation of a highly albuminous lymph into the tissue spaces, and leuco- or lympho-cytosis. Owing to the extreme vascularity of the iris and the peculiar distribution of the vessels, and to the looseness of the stroma, these generic features of inflammation produce special results. Thus, simple hyperæmia tends to cause the pupil to contract mechanically, on account of the radial disposition of the vessels. This is to some extent physiological, but is greatly increased under pathological conditions. The extreme vascularity and the looseness of the tissues causes an unusually large amount of exudation on the one hand and of swelling on the other. Owing to the greater albuminous content of the fluid its viscosity is increased so that it escapes into the anterior chamber and out of the anterior chamber by way of the filtration angle (*vide* p. 20) with greater difficulty. The iris, from being a partially wrung-out flat sponge, becomes a sponge full of sticky fluid. Hence its freedom of movement is greatly impaired, and the normal reactions become very sluggish or completely abolished. The fluid, too, contains deleterious substances which act as irritants; the nerve endings are stimulated so that the muscle fibres contract. In any case in which the sphincter and dilatator fibres are equally and uniformly stimulated the sphincter overcomes the dilatator, so that constriction of the pupil follows.

It is easy now to understand the chief signs of iritis. The pupil is constricted, partly owing to hyperæmia, partly to irritation; the edge of the pupil is markedly irregular. The reactions of the pupil are sluggish, partly owing to the same causes which induce constriction, partly to what may be termed water-logging. The latter condition causes an alteration in the appearance of the iris. The delicate iris pattern, instead of being clear and sharply defined, becomes blurred and indistinct ("muddy" iris). The colour undergoes considerable change, varying according to the condition of normal pigmentation. In fair people with little pigmentation, the blue iris becomes bluish or yellowish green; brown irides show less difference, but become greyish or yellowish brown. In any case comparison of the colour of the two irides will usually reveal some slight difference, for iritis is generally unilateral during the acute attack.

As a result of the change in colour and blurring of the iris pattern, the hyperæmia of the iris itself is not very obvious, but it manifests itself in circumcorneal ciliary congestion (*vide*

p. 83). This is most marked if the ciliary body is seriously involved, but the conjunctival vessels are also frequently somewhat engorged, so that care is necessary in distinguishing the condition from conjunctivitis. The secondary nature of the conjunctival congestion is shown by the relatively slight discharge: what discharge there is is chiefly lacrymal, never mucopurulent in the absence of actual conjunctivitis as a complication.

The iris is richly supplied with sensory nerves from the ophthalmic division of the fifth nerve. It is not surprising, therefore, that pain is a prominent symptom of acute iritis. It is not confined to the eye, though severe neuralgic pain is felt here, but is also referred to other branches of the first division of the fifth nerve, especially to the brow and parts supplied by the supraorbital and trochlear branches, but also to the cheeks and malar bone, and sometimes to the nose and teeth. It is worse at night.

The albuminous exudates escape slowly into the anterior chamber and mix with the normal aqueous. If the ciliary body is much involved the aqueous itself is plasmoid (*vide* p. 21). The aqueous often contains leucocytes and minute flakes of coagulated proteid, seldom fibrinous networks except in severe cases. It therefore becomes hazy, further interfering with a clear view of the iris and easily mistaken for haziness of the cornea, which is usually not involved. In very intense cases, especially of traumatic iritis with septic infection, large numbers of polymorphonuclear leucocytes are poured out; these sink to the bottom of the anterior chamber and form an hypopyon. Hypopyon is rare in simple iritis without perforation of the globe. Hyphæma, or blood in the anterior chamber, may also occur, but is rare in simple iritis.

The abnormal condition of the aqueous impairs the nutrition of the endothelium which covers the back of the cornea. The cells become sticky and may desquamate in places. The exudates tend to stick to the more affected spots, forming *keratic precipitates* ("keratitis punctata"). These are seldom present in simple iritis, but form an important feature of cyclitis, varying roughly with the amount of cyclitis present.

The more albuminous the aqueous the more viscous it becomes. This viscous fluid filters out of the anterior chamber by the filtration angle with difficulty. Hence there is a tendency for the fluid to be retained, so that the intraocular tension rises. The rise is minimal and of no serious import in cases of simple iritis—in fact, it is scarcely appreciable by

clinical methods. If, however, the ciliary body is much involved, the albuminous constituents of the aqueous are very markedly increased, and rise of tension may be so great as to endanger the sight, requiring special attention.

The exudates which are poured out by the iris and ciliary body are naturally most concentrated in their immediate neighbourhood. They cover the surface of the iris as a thin film and spread into, and often completely over, the pupillary area. In this manner the pupil may become "blocked" with exudates, a condition which very seriously impairs the sight of the eye. Moreover, the exudates tend to stick the iris down to the lens capsule, so that it becomes immovably fixed.

If the patient is seen in the early stages and atropine is instilled the pupil dilates, though not so readily as a normal pupil, on account of the water-logging of the iris. By continuous treatment the iris may be freed from the lens capsule and the pupil become completely dilated and circular.

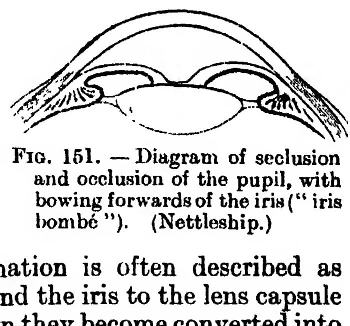


FIG. 151. — Diagram of seclusion and occlusion of the pupil, with bowing forwards of the iris ("iris bombé"). (Nettleship.)

The exudates, however, show a great tendency to become quickly organised. This is particularly seen in most cases of iritis, less frequently in cyclitis; when it is a very marked feature of the case the inflammation is often described as *plastic*. If the exudates which bind the iris to the lens capsule have not been already broken down they become converted into fibrous bands which atropine is wholly unable to rupture. Such firm adhesions of the pupillary margin to the lens capsule are called *posterior synechiæ* (συνέχειν, to hold together). When they are present a mydriatic causes only the intervening portions of the circle of the pupil to dilate and the pupil assumes a festooned appearance (Plate VI., Fig. 4; Fig. 155). Even in the absence of a mydriatic minute inspection will generally show irregularities of the pupillary margin due to the synechiæ. Such an irregular pupil is a sign of present or past iritis. For diagnostic purposes homatropine should be instilled and the result confirmed by the appearance of the dilated pupil (*vide* p. 261). Owing to the contraction of organising exudates upon the surface of the iris the retinal pigment epithelium may be pulled outwards over the pupillary margin. Patches of pigment are then seen in this situation

(ectropion of the uveal pigment). Posterior synechiæ show some predilection for the lower part of the pupil in the early stages, probably owing to gravitation of the plastic exudates.

In very severe cases of plastic iritis, or after recurrent attacks, the whole circle of the pupillary margin may become tied down to the lens capsule. The condition is called *annular* or *ring synechia* or *seclusio pupillæ* (Figs. 151, 154); it is one of great danger to the eye (*vide* p. 274). In similar cases,

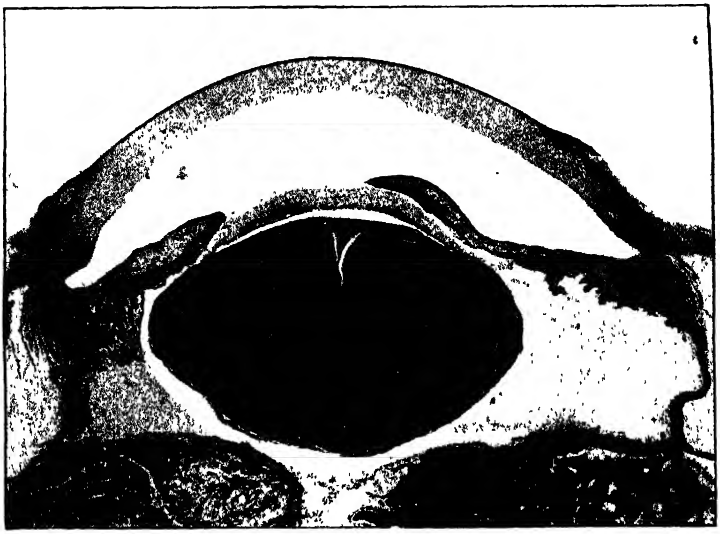


FIG. 152.—Total posterior synechia (× 7), from a case of plastic irido-cyclitis beginning to cause phthisis bulbi. The iris is completely adherent to the lens capsule, and the periphery is retracted. There is a delicate inflammatory pupillary membrane (occlusio pupillæ). There is also an anterior capsular cataract, due to inflammation; itcontains calcareous deposits, as shown by the patch of dark staining. The ciliary body is degenerated and detached from the sclerotic at the posterior part. The retina is completely detached and folded behind the lens.

especially if the case has been neglected and the pupil not well dilated by atropine at an early stage, the exudates in the pupillary area may also organise. A film of opaque fibrous tissue then fills the pupil; this condition is called blocked pupil, or *occlusio pupillæ* (Figs. 151, 154). If there has been much cyclitis the posterior chamber (Fig. 1) also becomes filled with exudates which may organise. They then tie down the whole of the back of the iris to the lens capsule; this condition

is called *total posterior synechia* (Fig. 152). It leads to retraction of the peripheral part of the iris, so that the anterior chamber becomes abnormally deep at the periphery, sometimes much deeper than in the centre.

Iritis is most frequently mistaken either for conjunctivitis or for acute glaucoma. The points which distinguish it from conjunctivitis will be gathered from what has already been described. The error of mistaking iritis for glaucoma is the most serious which can be made, more particularly because the treatment of the two conditions is diametrically opposed. Dilatation of the pupil with atropine, which is urgently necessary in iritis, is the worst possible treatment of glaucoma (*q.v.*). At the cost of some repetition, the distinguishing features will be given here.

(1) In iritis the pupil is smaller than normal and irregular; in glaucoma it is larger and oval, usually with the long axis vertical.

(2) In iritis the intraocular tension is scarcely appreciably raised unless much cyclitis is present; in glaucoma it is always appreciably raised, and is often very high.

These are the two chief objective signs. Cases occasionally arise which are doubtful even to the most experienced. A useful and harmless procedure which will almost invariably settle the question is the following: A drop of 2 per cent. euphthalmine or homatropine (*not* atropine) solution is instilled. As the pupil dilates, in iritis the irregularities are emphasised and synechiæ are almost always revealed; the tension is not appreciably affected by the mydriatic; in glaucoma the pupil probably dilates slowly but quite evenly, retaining its roundness; the tension is appreciably increased by the mydriatic. As soon as glaucoma is definitely diagnosed by this test eserine (1 per cent.) must be *immediately* instilled and repeated at intervals (*vide* p. 292). Atropine is never to be used for diagnostic purposes, since eserine is incapable of counteracting its mydriatic effect, and if the case were found to be one of glaucoma immediate operation would be imperative.

(3) The subjective symptoms differ in the two diseases. Vision is usually more impaired in glaucoma than in iritis. In acute glaucoma the onset of the pain is sudden, and it is so severe that it is frequently accompanied by vomiting.

It will be advisable again to enumerate the chief symptoms and signs of iritis. The subjective symptoms are: pain, of a neuralgic character, referred not only to the eye but also to the

supra-orbital region; dimness of vision, due to cloudiness of the aqueous, exudates in the pupillary area, &c. The objective signs are: constriction and irregularity of the pupil, unless a mydriatic has already been instilled, in which case the irregularity is emphasised by the presence of the posterior synechiæ. If the mydriatic is instilled early these synechiæ may break down so that the pupil again becomes quite round; in such cases spots of lymph or pigment upon the anterior capsule of the lens often leave permanent marks of old synechiæ, and form most valuable evidence of previous iritis (Fig. 153). It has already been pointed out (p. 4) that the posterior layer of the retinal pigment epithelium on the back of the iris is less firmly attached to the iris than the anterior. When a synechia breaks down some of the posterior layer often tears away and remains attached to the lens capsule; these pigmented spots never disappear entirely. They are easily distinguished from the congenital spots due to persistence of the pupillary membrane pigment (*vide* p. 278), and are valuable evidence of previous iritis. Discoloration or muddiness of the iris, whereby the iris pattern is masked; hyperæmia, manifesting itself chiefly as circumcorneal ciliary congestion; exudations, manifesting themselves either as more or less cloudiness of the aqueous or as solid deposits in the pupillary area and upon the iris: these are conspicuous signs of inflammation of the iris.

FIG. 153.—Spots of pigment on the lens capsule, left by posterior synechiæ which have broken down. (Nettleship.)

The course of iritis varies with its intensity. Even the slighter acute cases take three or more weeks before inflammation entirely subsides. The best sign is the prompt action of atropine, for in the worst cases it has little or no effect. Improvement is shown by good dilatation, diminution of injection and pain. In the chronic cases the ciliary body is almost always more seriously involved; the condition is one of iridocyclitis. The inflammatory signs are less, but diminution of vision is progressive, and the disease not infrequently lasts for years.

One of the most characteristic features of iritis and cyclitis is the great tendency to relapse. It depends, not upon the synechiæ, as was once taught, but upon the constitutional cause of the disease. Each fresh attack runs a similar, though usually less severe, course, often leaving fresh traces and increased impairment of vision.

Complete resolution may occur in slight cases treated early

and suitably. The exudates become absorbed ; the synechiæ break down, leaving only such slight traces that vision may be perfect. Comparatively slight cases may, however, leave very serious results if they are improperly treated, and in severe cases these are the rule. Most of the evil results are attributable to neglect of or impossibility of early dilatation of the pupil, which causes permanence of the posterior synechiæ. If these are few, no special injury or impairment of sight follows,

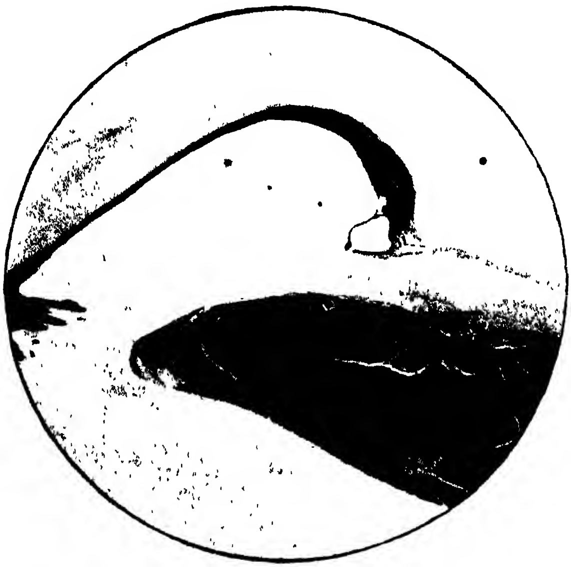


FIG. 154.—Iris bombé, with very broad peripheral anterior synechia, annular posterior synechia (seclusio pupillæ), and inflammatory pupillary membrane (occlusio pupillæ). There is also an anterior capsular cataract.

but future attacks are more likely to result in an increased number or in ring synechia.

Ring synechia, or seclusio pupillæ, is one of the worst sequels of the disease, since, if unrelieved, it inevitably leads to secondary glaucoma and destruction of sight. Owing to the complete shutting off of the pupil the aqueous is unable to pass forwards into the anterior chamber, the pigment epithelium forming a non-permeable membrane. It therefore collects behind the iris, which becomes bowed forwards like a sail, a condition which is called *iris bombé* (Fig. 154). Regarded from in front, the anterior chamber is seen to be funnel-shaped,

deepest in the centre and shallowest at the periphery. The filtration angle is obliterated by the adhesion of the iris to the cornea and sclera at the periphery (peripheral anterior synechia). Hence the fluid is retained within the eye and the intraocular tension rises (*vide* p. 281).

Organisation of the exudates in the pupillary area leads to the formation of an inflammatory pupillary membrane or occlusio pupillæ. This interferes directly with the transmission of light rays and is often associated with ring synechia, with or without total posterior synechia (*vide* p. 261). In such cases there is generally plastic cyclitis and the eye is irretrievably affected.

Repeated attacks of iritis lead to atrophy of the iris, which becomes dirty grey, like felt or blotting paper. Red streaks often mark the site of permanently dilated vessels, usually of new formation and therefore not necessarily radial in direction. The pupillary margin is thin and frayed: the reactions are diminished.

FIG 155. — Plastic iritis with nodules in the angle of the anterior chamber. (Nettleship, from a drawing by Holmes Spicer.) The patient was not syphilitic.

Varieties of Iritis. The varieties of iritis are usually divided into primary and secondary, the latter being those which are due to extension of inflammation from some other part of the eye, usually the cornea. The primary iritides are due to some general dyscrasia, though it is not always possible to determine its exact nature (so-called idiopathic iritis). The most undoubted cause is certainly syphilis; other causes are gonorrhœa, tubercle, and diabetes. There is both clinical and experimental evidence that certain strains of streptococci have a specific selectivity for the tissues of the uveal tract. Alleged causes are gout, "rheumatism," acute exanthemata, &c. Finally, iritis is usually an important element of sympathetic ophthalmia (*q.v.*).

Syphilitic Iritis manifests itself in two forms. Syphilis is the commonest cause of simple plastic iritis, which occurs in the secondary stage, soon after the skin eruptions, usually within the first year after infection, but not before the third month. There may be nothing characteristic about this form of iritis, or nodules may occur upon the iris (*vide infra*). Syphilitic iritis lasts two to eight weeks and does not usually recur, thus differing from the "rheumatic" and gonorrhœal forms.

Iritis used to occur in at least 3—4 per cent. of syphilitics, and of cases of iritis syphilis accounted for at least 25—30 per cent., but syphilitic iritis appears to be less common than formerly, probably owing to earlier and more thorough anti-syphilitic treatment. Syphilitic iritis is generally unilateral, but the second eye becomes affected sooner or later in about a quarter of the cases. Iritis due to other causes is unilateral in only about 10 per cent. Syphilitic iritis attacks males more than females, and three-fourths of the cases are between twenty and forty years of age. The Wassermann reaction is of great value in settling the diagnosis. The spirochæte has been found in the aqueous.

Simple plastic iritis also occurs in congenital syphilis, usually as an accompaniment of interstitial keratitis (*q.v.*). It also occurs in very young babies with congenital syphilis, without any corneal complication, but usually with large nodules or gummata on the iris. This is not common, but is probably the only cause of iritis in very young children apart from direct injury. It is sometimes seen so soon after birth that almost certainly in these cases it commences as an intrauterine inflammation. The average age of onset is five to six months. It is commoner in females than in males, and is unilateral in about half the cases (Hutchinson.)

FIG. 156.—Nodules occurring in the secondary stage of syphilis, situated at the pupillary border of the iris. (Nettleship, from a drawing by W. G. Laws.)

The iris also becomes inflamed in some cases of acquired syphilis late in the secondary or rarely during the tertiary stage. These cases are characterised by the formation of yellowish red nodules near the pupillary and ciliary borders of the iris, but not in the intermediate region. The nodules are usually multiple and vary in size from that of a pin's head upwards (Fig. 156). It has been customary to consider these nodules either condylomata or gummata, according to the stage of the complaint. There is no good ground, either clinical or pathological, for the distinction, and the term *gummatous iritis* may be used for all these cases. There is generally much exudation in gummatous iritis, and broad synechiæ are formed. The nodules are liable to be mistaken for tubercle (*q.v.*) or sarcoma (*q.v.*); the absence of iritis and the presence of only a single nodule usually distinguishes the

latter condition, which, moreover, is extremely rare. Gummatous iritis may rarely extend to the corneo-sclera at the angle of the anterior chamber and lead to perforation of the globe.

The sites of previous gummatous deposits in the iris are marked by depigmentation of the stroma, probably owing to stretching. Whitish spots in the ciliary region of the iris, especially near the crypts, occur in the early stages of syphilitic infection without previous iritis. Syphilitic degeneration of the vessels causes thickening of the coats, sometimes making them appear as white lines. Atrophy of the muscle fibres, especially of the sphincter, also occurs, and a circle of atrophic patches near the pupillary border is strong evidence of syphilis.

Vision is usually impaired permanently in syphilitic iritis, and the prognosis is worse in the gummatous type. Specific iritis is indeed a sign of severe syphilis ; Trousseau found that thirty-four out of forty patients ultimately developed grave sequelæ such as tabes and general paralysis.

Gonorrhœal Iritis is probably more common than is generally supposed. It occurs especially in those cases which have gonorrhœal "rheumatism" and seldom supervenes until after one attack of arthritis, usually in the knees. It tends to recur, often during the onset of gleet or arthritis, and often many years after the first attack. There is little doubt that it is a metastatic infection. The patients are almost always men, and as a rule both eyes are affected, though not at the same time. Extension to the ciliary body may be indicated by fine vitreous opacities, but involvement of the choroid is very rare. Another more characteristic form may occur during the acute attack. The exudates into the anterior chamber then have a peculiar gelatinous appearance and a greenish-grey colour which is characteristic.

"*Rheumatic*" *Iritis*. In patients with iritis, in whom no specific or gonorrhœal taint can be discovered, a history of rheumatic pains in the muscles and joints can often be elicited. Iritis seldom, if ever, accompanies an attack of acute rheumatism, and only rarely can a history of such an attack be obtained. The patients are often gouty or have rheumatoid arthritis. What the pathological relationship of the iritis is to these complaints must remain a matter of conjecture until their ætiology is placed beyond dispute. It is most probable that both the "muscular rheumatism" and the iritis are due to a common cause, toxins derived from a septic focus in some part of the body, *e.g.*, mouth, nasal sinuses, intestinal tract,

&c. The iritis in these cases is usually a plastic iritis of moderate severity. It often attacks both eyes and shows a very marked tendency to recur, and the recurrence seems to bear a direct relationship to the recurrent attacks of pain or arthritis. There is often an unusual amount of conjunctival and episcleral congestion. Iritis in an elderly patient is likely to be gouty, often starting suddenly in the night and sometimes ushering in an acute attack of gouty arthritis.

Diabetic Iritis is rare: it may be due to deficiency in riboflavin (*v.* p. 249). It shows a marked liability to the formation of new or enlarged vessels in the iris (*rubeosis iridis*), with the formation of plastic exudates and occasionally an hypopyon. On the whole it runs a favourable course.

Tuberculous Iritis occurs in a miliary and a conglomerate or solitary form. In the former there is usually a yellowish white nodule surrounded by numerous smaller satellites; there is the same tendency as in gummatous iritis for the nodules to be near the pupillary or ciliary margins. In the earliest stages the nodules are minute, greyish, and translucent. There are often spots of "k.p." on the back of the cornea, indicating involvement of the ciliary body. Hyphæma is not uncommon; and pseudo-hypopyon, composed of caseating tuberculous material, may occur. In conglomerate tubercle there is a yellowish white tumour, though smaller satellites may be present. The nodules contain giant cells. There is usually less general iritis than in the gummatous form, but there is almost always some. The condition may be mistaken for gummatous iritis or for sarcoma. The absence of specific history, a negative Wassermann reaction, the failure of anti-syphilitic treatment, and the age of the patients—children or young adults—are features distinguishing it from gummatous iritis. The presence of satellites, the usual, but not invariable absence of visible vessels upon the surface of the nodules, the age of the patient, and the presence of iritis distinguish it from sarcoma. The diagnosis may be extremely difficult, but the great rarity of sarcoma of the iris should be borne in mind. Simple iritis is said to be sometimes tuberculous.

In conglomerate tubercle of the iris the corneo-sclera at the angle of the anterior chamber almost invariably becomes ultimately eroded and involved. The wall of the globe is thus weakened and eventually gives way. The tuberculous mass then grows rapidly through the perforation, and a large portion of the iris may become prolapsed. In this manner the eye is inevitably lost.

von Pirquet's cutaneous reaction may be applied to doubtful cases. A positive result is of little value except in children, but a negative result eliminates the diagnosis of tubercle with a fair degree of certainty. Wolff-Eisner or Calmette's conjunctival test should not be used on account of the danger of severe reaction. Subcutaneous injection of Koch's old tuberculin gives a characteristic rise in temperature, &c., in the presence of tubercle, but there is no proof that the ocular lesion is the cause of the reaction, and the test is not free from danger, due to violent local reaction.

Brucellosis (*Syn.* Undulant Fever). Infection by the three strains of the Brucella (abortus, sius or meletensis) is widespread throughout the world and among the many sites of its manifestations the eye may be affected. A keratitis and optic neuritis is rare; a uveitis of a chronic granulomatous habit is commoner. The disease is prone to relapse and diagnosis can only be suggested from other forms of chronic irido-cyclitis by an agglutination test, a cutaneous test, or an opsonocytophagic test.

Treatment. Dilatation of the pupil with atropine and hot applications are the essentials of local treatment. Atropine acts in three ways: (1) by keeping the iris and ciliary body at rest; (2) by diminishing hyperæmia; (3) by breaking down posterior synechiæ and preventing the formation of fresh ones. It may be used in the form of drops of a 1 per cent. solution or as an ointment of the same strength. I prefer the ointment for the following reasons: (1) its action is more continuous; (2) it is easier to apply, since it usually works into the eye even if only rubbed along the lashes; (3) it is less likely to cause symptoms of poisoning, which are not uncommon with the drops in children. Symptoms of poisoning—dryness of the throat, flushing of the face, delirium, &c.—are due to the excess of solution—often considerable in unskilful hands—passing down the nasal duct into the nose and throat. The dose administered in this manner is never lethal.

Atropine should be pushed in the early stages, best by frequency of application rather than increased strength. Every four hours is usually sufficient. When the pupil is well dilated, two or three times a day suffices. If atropine irritation ensues, hyoscine, scopolamine, or duboisine should be substituted. Dionin, 5—10 per cent., may be used in conjunction with the mydriatic.

A very powerful mydriatic effect is obtained by the subcon-

junctival injection of 5 minims of mydricain (*vide* p. 694), a mixture of atropine, cocaine, and suprarenin.

Hot applications are extremely grateful to the patient, diminishing the pain, and are of therapeutic service in encouraging a more vigorous blood and lymph flow. Hot fomentations and bathings may be used, but dry heat applied to the surface of the closed lids has the same effect. This may be done by means of medical diathermy (300 to 600 milliampères for five minutes) or an electric heater, which is bandaged over a pad of wool, previously well heated and applied to the eye. By this means the heat is considerable and continuous, with a minimum of trouble and discomfort.

In very severe cases, or when the pupil does not readily respond to atropine, depletion of blood from the temple should be resorted to. The best method is by two or three leeches applied a short distance outside the external canthus. They should not be too far from the eye, nor too near, for in the latter case much œdema of the lids may follow. Heurteloup's artificial leech may be used, but is not so efficacious, since the leech extract diminishing the clotting capacity of the blood has a beneficial effect.

If the pain is very intense a hypodermic injection of morphia may be given. Aspirin is very useful in relieving pain.

General treatment should be commenced by a saline purge, and the bowels must be kept freely open throughout the acute stage.

In other respects the general treatment depends upon the cause. In syphilitic iritis the patient is rapidly got under the influence of mercury, best by inunctions or the intramuscular injection of Lambkin's cream. N.A.B. injections cause rapid improvement, but these cases also respond well to mercury. These drugs are most effectual in the cases occurring during the secondary stage, but should also be used in the gummatous form. Here they should be supplemented by iodides, but these must not be given simultaneously with injections of metallic mercury. The infantile form of acute syphilitic iritis responds rapidly to mercury, but neither drug is very efficacious in the congenital type, and in this, as well as in the later stages of the other forms, general tonic treatment is indicated.

In gonorrhœal iritis an intensive course of sulphonamides in full doses (sulphathiazole, sulphadiazine) should be started at once (*vide* p. 695). In chronic or recurrent cases gonococcic vaccine sometimes produces good results, and massage of the

prostate, though it may cause a temporary exacerbation, helps to remove the source of the trouble.

In other forms of iritis an exhaustive search should be made for any septic focus in the body and the result anticipated, particularly if a streptococcal origin is suspected, by a course of one of the sulphonamides (*vide* p. 695). If the response is not good, or where no satisfactory cause can be found, it is usual to order salicylic preparations, and they appear to do good, not only in these, but also in gonorrhœal and diabetic iritis.

In the convalescent stage smoked glasses are ordered—for both eyes, especially on account of the consensual reaction to light. Atropine, or its equivalent, should be continued for at least ten days or a fortnight after the eye appears to be quiet, otherwise a relapse is very likely to occur.

Tuberculous iritis is treated by the same local applications as other forms. The usual constitutional treatment must be pushed. Improvement and even complete resolution have been recorded from the use of tuberculin injections, but they should be used cautiously, commencing with very small doses, since a violent reaction may have a serious effect upon the eye. The dose is very gradually increased.

Some authors consider that tuberculous iritis is generally the primary manifestation of the disease in the body. They therefore advocate the removal of the eye as soon as the diagnosis is made, in order that the danger of extension of the disease to other parts of the body may be minimised. If perforation of the globe has taken place and the eye is irretrievably lost immediate excision should be urged, but in other cases ordinary treatment, supplemented by tuberculin, should be persisted in. Good results have undoubtedly been obtained by the use of tuberculin, and it should be given a persevering trial; nevertheless it often fails to ameliorate the condition.

In brucellosis, apart from the usual local treatment, sulphonamides may be given (*vide* p. 695). Vaccines have also been employed, but in general treatment is unsatisfactory.

Treatment of Sequelæ and Complications. For the treatment of coincident cyclitis see p. 275.

Firm posterior synechiæ can sometimes be broken down by placing a small particle of solid atropine in the conjunctival sac. Care must be taken to prevent the dissolved atropine from passing down the nasal duct by pressure with the finger

upon the lacrymal sac by the patient himself, but the surgeon must see that the pressure is rightly applied and kept up. Alternatively, and usually more effectively, a subconjunctival injection of midricain may be given and repeated if necessary (*vide* p. 694).

Annular synechia demands an iridectomy in all cases in order to restore communication between the anterior and posterior chambers, and thus avoid the supervention of secondary glaucoma (*vide* p. 282). In some cases it is necessary or advisable to be content with making a puncture or transfixion of the iris by a broad needle. No operative procedure of this kind must be undertaken during an acute, or even the slightest attack of iritis, if it can be possibly avoided, since the traumatic iritis set up will frustrate the object of the operation by filling the coloboma with exudates, and may even cause the loss of the eye. It is best, if possible, to forestall a ring synechia by performing the iridectomy before the adhesion extends round the whole circle. This can often be done, because operable ring synechia, *i.e.*, ring synechia without total synechia, is frequently the result of recurrent attacks of quiet or almost painless iritis, during each of which more of the circle is involved. The iridectomy is performed during a quiescent interval. It is often difficult to get a good coloboma owing to the atrophy and friability of the iris and the firmness of the adhesions. Hæmorrhage is common, and the hyphæma takes longer than usual to be absorbed. If for any reason operation on such an eye appears to be specially risky, particularly if it is an only seeing eye, the pupil should be kept continuously under atropine (0·5 per cent.) instilled once a day. Iridectomy sometimes has a beneficial effect on recurrent iritis, but should not be done without special indications until all other non-operative measures have failed. The presence of "k.p." should generally be regarded as a contra-indication to intraocular operation in the absence of dangerously high intraocular pressure.

Total posterior synechia can seldom be operated upon with success. Iridectomy is seldom possible, and the only procedure which can be adopted is extraction of the lens by a specially devised operation. Iridectomy or iridotomy may be possible if the lens is shrunken.

Cyclitis has already been referred to incidentally. In the severe *plastic* form the exudates from the ciliary body pass into the anterior chamber directly from that part which forms a boundary of the chamber (Fig. 2), and indirectly by passing

forwards through the pupil. When they organise they not only cause total posterior synechia, but also surround the lens and extend throughout the vitreous. Behind the lens they form a transverse membrane or cyclitic membrane. Strands of fibrous tissue are formed in the vitreous. They become anchored to the retina in various places, and by their subsequent contraction often lead to detachment of the retina. The exudates which organise upon the surface of the ciliary body cause the destruction of the ciliary processes, which results in abolishing or seriously diminishing the secretion of aqueous. Hence the intraocular tension becomes lowered (*hypotony*) and

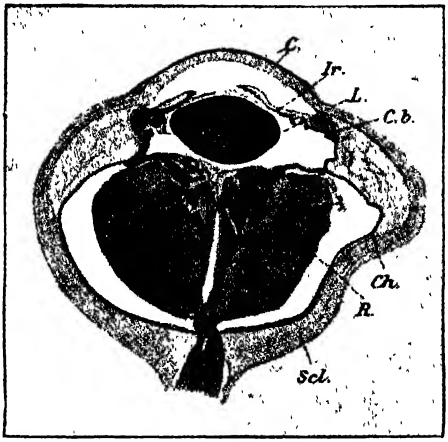


FIG. 157.—Phthisis bulbi, due to irido-cyclitis (× 3). *C*, cornea; *Ir.*, iris; *L*, lens; *C.b.*, ciliary body; *Ch.*, choroid; *R*, retina, detached and folded up behind lens, separated from choroid by albuminous coagulum; *Scl.*, sclerotic.

the eye is quite soft to the touch. The walls of the globe fall in, and the eye becomes shrunken and quadrilateral in shape owing to pressure by the recti muscles—*phthisis bulbi* (φθίνειν, to waste) (Fig. 157). After this has occurred degenerative changes supervene, and the choroid becomes converted after months or years into bone.

Chronic Irido-cyclitis (*Syns.—Simple Cyclitis,* "*Serous Iritis*") is an extremely insidious and chronic disease, characterised by diminution of vision with slightly marked physical signs. In severe cases there is ciliary congestion, tenderness on pressure over the ciliary region, deep anterior chamber, precipitates ("keratitis punctata") on the back of the cornea, and dust-like opacities in the vitreous. Posterior synechiæ

are not a conspicuous feature, but are liable to be formed slowly and insidiously. The tension is usually slightly raised in the earlier stages, lowered in the later. There may be œdema of the upper lid and neuralgic pain in the eye and brow. There is sometimes myopia, owing to irritation of the ciliary muscle.

The *keratic precipitates* ("k.p.") (Fig. 158) consist of lymphocytes which are deposited from the aqueous upon the back of the cornea and stick there. They may contain pigment granules, showing their origin from the uveal tract (pigmented k.p.). In the most characteristic form they are scattered over a triangular area of the lower part of the cornea, the smaller spots being above, the larger below (Fig. 158). This arrangement is due to convection currents and gravitation of the particles towards the bottom of the anterior chamber, combined with the perpetual movements of the eye, which are mostly in the horizontal direction. The typical arrangement is often but not always observed. More commonly a few isolated spots are seen scattered irregularly over the lower part of the cornea. They require great care in examination for their discovery (*vide* p. 87), and their importance cannot be over-estimated. The smaller spots frequently coalesce, forming small plaques, which gradually become more translucent ("mutton-fat k.p."). Precipitates are generally sharply defined and can thus be distinguished from opacities in the deeper layers of the cornea. They are more likely to contain pigment when the iris is brown, but "pigmented k.p." also occurs with blue or grey irides if the inflammation lasts a long time. Pigmentation therefore may give some indication of the duration of the disease. The pigment persists almost indefinitely. Similar precipitates are rarely seen upon the anterior capsule of the lens, but the leucocytes do not readily stick here owing to the smooth surface, devoid of endothelium.

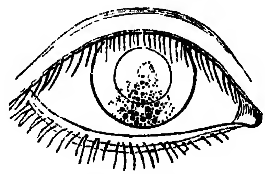


FIG. 158. — Typical arrangement of the spots on the back of the cornea in "keratitis punctata" (k.p.). (Nettleship, from a sketch by Dr. Herringham.)

The vitreous opacities are of the same nature, viz., wandering leucocytes, but many are also probably minute particles of albuminous exudate. Their mobility in the vitreous shows that the consistency of this substance has undergone change, sometimes amounting to complete fluidity, due to defective nutrition.

The increased depth of the anterior chamber is an important sign, not easily explained. It is undoubtedly connected with the deficient filtration of the plasmoid aqueous through the angle, which, together with dilatation of capillaries produced by the secretion of histamine-like substances and axon reflexes, also accounts for the rise in tension. The peripheral part of the anterior chamber is often particularly deep, even deeper than the central: this is, however, more marked in the later stages of plastic cyclitis, when it is due to mechanical retraction of the iris from organisation of exudates in the posterior chamber.

In severe or prolonged cases the deeper layers of the cornea may become infiltrated, as in interstitial keratitis, though seldom to the same extent. This is specially liable to occur in tuberculous cases, and in these there are often minute greyish nodules on the surface of the iris. These should be carefully looked for, as they are of considerable diagnostic significance, and the prognosis is correspondingly grave. Tiny translucent nodules are sometimes seen, even in early stages, at the pupillary margin of the iris. These cases seem invariably to go from bad to worse in spite of treatment. They are probably tuberculous.

In the slightest and most insidious cases of irido-cyclitis the symptoms and physical signs are minimal. Considerable diminution of vision without obvious cause should always excite apprehension, and the cornea should be most carefully explored by oblique illumination with magnification by the loupe, as well as by the direct method with a strong convex lens. A few spots of "k.p." are decisive proof of cyclitis, and may be the sole physical sign. Change in the colour of the iris, due to atrophy, is an important sign, since it may at once attract attention, especially if the normal eye has a brown iris. It indicates, however, a late stage of the disease.

Chronic irido-cyclitis occurs under similar conditions to simple iritis and is commoner in women than in men. Syphilis or tubercle may be the cause. It is probably always due to some form of toxæmia or bacterial metastasis, and a careful search should be made for a septic focus in some part of the body. It is most frequently found in the mouth—pyorrhœa alveolaris—in the nose and accessory nasal sinuses, or, especially in women, in the genital tract. The frequency of streptococcal bacteriæmia associated with pyrrhœa has been conclusively proved, the streptococci being usually of the

viridans type—10 per cent. of cases ; 70 per cent. immediately after extraction of teeth (Okell and S. D. Elliott.) Very often no such source of toxins can be demonstrated, but the patients are usually of a debilitated type. There is often constipation, and it is not improbable that the intestinal tract is a frequent source of the toxins.

The disease is generally very chronic and liable to exacerbations with gradual and insidious formation of posterior synechiæ. Vision is greatly diminished during the more acute stage, and recovers considerably in the intervals, but each recurrent attack leaves more and more permanent defect. There is usually increased tension during the more acute stages, and this may be so great as rapidly to abolish vision unless relieved. The eye may finally become soft and tender, and enter into the condition of phthisis bulbi, but this occurs only after many years in simple cases of irido-cyclitis. Less serious cases, however, not uncommonly occur, especially when the septic focus, *e.g.*, pyorrhœa alveolaris, is discovered and is amenable to radical treatment.

Treatment of irido-cyclitis is essentially the same as that of iritis, but special attention must be directed to any septic foci which may be found and to the general health. Septic foci such as pyorrhœa alveolaris, nasal, genital, or urinary sepsis, furunculosis, &c., must be radically treated when possible. If there is extensive pyorrhœa only four or five teeth should be removed at a time, since absorption of toxins from the gums may cause a severe exacerbation of the cyclitis and even alarming shock. In some cases an autogenous vaccine has produced rapid cure, but more often it fails. The cases often drag on indefinitely, with occasional exacerbations. During the more acute phases energetic treatment with atropine, hot bathings or the electric heater, and if necessary blisters or leeches, is indicated, together with the administration of sulphonamides (*vide* p. 695). Small doses of calomel (gr. ¼, three times a day) or salicylates do good in many cases of obscure ætiology, probably by acting as intestinal disinfectants. Iodides help to cause absorption of vitreous and other exudates. Hexamine has been recommended on theoretical grounds, but has proved disappointing. In severe cases the patient should be kept in bed and submitted to mercurial inunctions or baths, which often do good in cases in which there is no specific history. Diaphoresis by vapour baths and hypodermic injections of pilocarpine may be tried in intractable cases, which form the majority. Cases of syphilitic or tuber-

culous origin, of course, require the appropriate methods of treatment, but tuberculin should be used with caution (*vide* p. 270). Some cases, probably tuberculous, show marked improvement after a course of ultra-violet radiation of the skin ; the eyes should be carefully protected by suitable glasses (*vide* p. 189) during the exposures. Other cases are improved by protein shock (*vide* p. 697).

If the intraocular tension is raised seriously, so that there is danger to the sight of the eye from this cause, it must be relieved by paracentesis (*vide* p. 209). This usually has only a transitory effect, and may require to be repeated every two or three days. It is theoretically unsatisfactory, since the comparatively sudden reduction of the intraocular pressure to zero causes dilatation of the ciliary vessels and allows the passage of a lymph which is even more albuminous than that which has been let out. It should therefore not be resorted to unless imperatively indicated. On the other hand, the final result is often very satisfactory, probably because the rapid flow of lymph flushes out the secretory channels and carries away endothelial and epithelial débris and stagnant toxins. If repeated paracentesis fails to relieve the tension, an iridectomy may do good ; it may be necessary to do a Herbert's sclerotomy or an anterior sclerectomy (trephining), but this should only be done as a last resource, since the results are usually very disappointing.

During the intervals between exacerbations the pupils should be kept moderately dilated with weak (0·5 per cent.) atropine. The patient should have plenty of fresh air, good diet, and tonics.

If the eye becomes useless, shrunken, and painful it may be necessary to excise it.

Plastic Irido-cyclitis. The pathology of this condition has already been described incidentally. In it the signs of irido-cyclitis in general are increased. The cyclitic membrane behind the lens may be seen with the ophthalmoscope or even by oblique illumination. In young children the condition forms one type of pseudo-glioma (Chap. XIX.). In the later stages the degenerative changes in the ciliary body prevent it from fulfilling its functions of supplying the eye with lymph and nutriment. The vitreous suffers first, becoming fluid, and later the lens, which becomes opaque. Finally the eye shrinks (phthisis bulbi).

Treatment is the same as for chronic irido-cyclitis. The blind, shrunken globe is often painful and a continual source

of annoyance to the patient. It should, in these circumstances, be excised.

Gumma of the Ciliary Body causes an intense acute plastic irido-cyclitis, with severe iritis, much exudation into the anterior chamber, often deep infiltration of the cornea and usually great pain. It is a rare complication of syphilis, not confined to the tertiary stage. It varies in the severity of the symptoms and the rate of progress. It is only to be diagnosed clinically with certainty when the inflammation extends into the sclerotic (*vide* p. 254). If it does not respond to active anti-syphilitic treatment, the eye eventually shrinks.

Tubercle of the Ciliary Body occurs with tubercle of the iris and of the choroid, and is usually only to be inferred clinically rather than definitely diagnosed. As already mentioned, the tubercle bacillus may account for some cases of chronic cyclitis.

Uveoparotitis (*Syn.—Heerfordt's Disease*) is a chronic bilateral parotitis and uveitis, generally occurring between ten and thirty years of age. A low grade fever, sometimes accompanied by a rash like erythema nodosum, precedes or follows the swelling of the parotid. There is frequently paralysis of the viith nerve, and other signs of peripheral neuritis, *e.g.*, ptosis, diplopia, recurrent laryngeal paresis, occur. The parotid swellings may last for six weeks to two years, but gradually subside. Evidence of tubercle has been found in all cases examined histologically.

An allied condition is *Boeck's sarcoidosis*, a low grade uveitis associated with nodules in the iris, cornea, skin, &c.

Secondary Iritis. See Chap. XXI.
Purulent Irido-cyclitis. See Chap. XXI.
Sympathetic Irido-cyclitis. See Chap. XXI.

Degenerative Changes in the Iris.

Depigmentation of the iris is seen in old people, and examination with the slit-lamp has shown that disintegration of the iridic pigment is a constant senile phenomenon. Depigmentation of the pupillary margin is common and may occur in the form of small triangular patches or radial fissures. Irregular lacunæ in the retinal pigment may often be seen by transillumination, either by the slit-lamp or by contact illumination. Atrophy of the stroma, especially near the pupil, is also common, and the pupillary border may be frayed out and very irregular, independent of inflammatory changes.

Congenital Abnormalities of the Iris.

One iris may have a different colour from the other, or parts of the same iris, usually a sector, may differ in colour from the remainder. Both conditions are known as *heterochromia iridis*. The blue iris is due to the absence of pigment in the iris stroma, the pigment in the retinal epithelium being seen through the translucent stroma. The eye with the lighter iris seems to be specially prone to iridocyclitis. *Many of these cases are due to wrong diagnosis, the lightness of the iris being due to degeneration following* iridocyclitis; but this explanation does not account for all the cases (Fuchs).

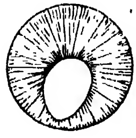


Fig. 159. — Congenital coloboma of the iris.

Irides often have patches of brown pigmentation; these benign *melanomata* are due to abnormal groups of retinal pigment epithelium lying in the posterior layers of the stroma.

The pupil is normally slightly to the inner side of the centre of the cornea. In some cases it is considerably displaced, usually also to the nasal side—*corectopia* (κόρη, pupil, ἐκ, out of, τόπος, place). Rarely there are other holes in the iris besides the pupil—*polycoria*.

The iris may be apparently absent—*aniridia* or *irideremia*. Anatomical investigation has shown that there is always a narrow rim persistent at the ciliary border, but it is hidden from view during life by the sclerotic. On examination the ciliary processes and the suspensory ligament of the lens can be seen. Aniridia is usually bilateral. There is a tendency for secondary glaucoma to develop in these eyes, a remnant of the iris blocking the angle of the anterior chamber.

There may be a gap in the iris, usually pear-shaped or like a Gothic arch, continuous with the pupil and extending towards, but not always as far as, the ciliary border. It is called a *congenital coloboma* (κολόβωμα, mutilation) of the iris (Fig. 159). It is usually downwards or down and slightly in, corresponding with the position of the fœtal, so-called choroidal cleft: such a coloboma is called typical. Colobomata are found in other directions, and are then atypical. Typical coloboma of the iris is often associated with typical coloboma of the choroid (*q.v.*), and in some cases with coloboma of the lens. It is one of the commonest congenital malformations of the eye.

Persistent Pupillary Membrane is due to persistence of part

of the anterior vascular sheath of the lens, a fœtal structure which normally disappears shortly before birth. Fine threads stretch across the pupil, or may be anchored down to the lens capsule (Plate VI., Fig. 3). They are distinguished from post-inflammatory synechiæ in always coming from the anterior surface of the iris just outside the pupillary margin, *i.e.*, from the position of the corona or circulus iridis minor. Such tags are of frequent occurrence and are of no pathological importance. They are commonest in babies and probably undergo further absorption as age advances, but many persist permanently. *Examination with the slit-lamp shows that minute* remnants of the pupillary membrane are very common even in adults.

The fœtal pupillary membrane consists of a network of minute blood vessels supported by a very delicate stroma which contains pigment cells. Sometimes the pigment is left on the lens surface and persists. It forms a stippling of very fine brown dots scattered over a circular area 5 or 6 mm. in diameter in the centre of the pupil. These spots are distinguished from pigment spots left by posterior synechiæ which have broken down (*vide* p. 262) in being much smaller, stellate in shape when magnified under the slit-lamp, much more numerous and very regularly arranged, and also by the absence of any concomitant signs of iritis. They do not usually interfere appreciably with vision.

Tumours and Cysts of the Iris.

Tumours of the Iris and Ciliary Body. See Chap. XX.

Cysts of the Iris. *Serous cysts* of the iris sometimes occur, and are due to closure of iris crypts, with retention of fluid. *Cysts of the retinal epithelium* occur, due to accumulation of fluid between the two layers of retinal epithelium. They look like a bombé iris limited to parts of the iris—a limitation which is impossible in the case of true bombé iris (*q.v.*). In these cases the posterior layer of epithelium is often adherent to the lens. *Implantation of epithelium* on the iris sometimes occurs after perforating wounds or operations, giving rise to pearl cysts or cholesteatomata. The epithelium may spread over the iris and line the whole anterior chamber, causing glaucoma. Many such cases are not true implantation cysts, but are due to downgrowth of epithelium from the conjunctiva occurring in badly healing wounds. Eyelashes are sometimes carried into the anterior chamber in perforating wounds and lodge upon the iris.

CHAPTER XIV

Glaucoma

GLAUCOMA is a symptomatic condition, not a disease *sui generis*. The characteristic physical sign is increased intraocular pressure. It will be clear from the description of the mechanism whereby the normal intraocular pressure is maintained (*vide* p. 14) that increase may be due either to (1) increased production of lymph associated with normal or diminished outflow, or to (2) diminished outflow associated with normal or increased inflow. The factors which cause increased production of aqueous, such as increased permeability of the capillaries, brought about, perhaps, by the presence of histamine-like substances and by axon reflexes, are probably particularly effective in producing acute rises in the ocular tension, while slow or chronic conditions of raised tension are probably more usually due to defective outflow. It has been suggested that in some cases the vitreous plays a preponderant part, but the rationale must remain speculative until our knowledge of the properties of the vitreous gel is further elucidated.

Two great classes of cases in which the tension is pathologically increased can be distinguished, viz., (1) those in which the tension is only moderately increased, in which the anterior chamber is deep, and in which there are more or less definite signs of inflammation of the ciliary body (Chap. XIII.); and (2) those in which all grades of increased tension are met with, in which the anterior chamber is shallow, and in which, though there may be very evident signs of congestion and irritation, any definite signs of ciliary inflammation are either absent or secondary in onset. It is well to keep these two groups quite separate, since their pathogenesis is different and the differences in clinical course and treatment are marked. The term glaucoma should be limited to the second group.

True glaucoma may be conveniently divided into two forms, primary and secondary. Since the pathology of secondary glaucoma has been fairly well elucidated and throws some light upon that of primary, the former will be considered first.

Secondary Glaucoma. We have seen that the increased

PLATE II

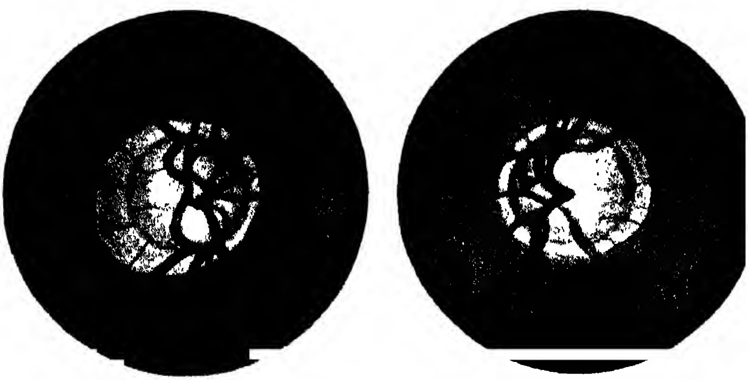


FIG. 2.—R. and L. cupped discs: primary glaucoma.

PLATE IX.

FIG. 1.—Disseminated choroiditis.

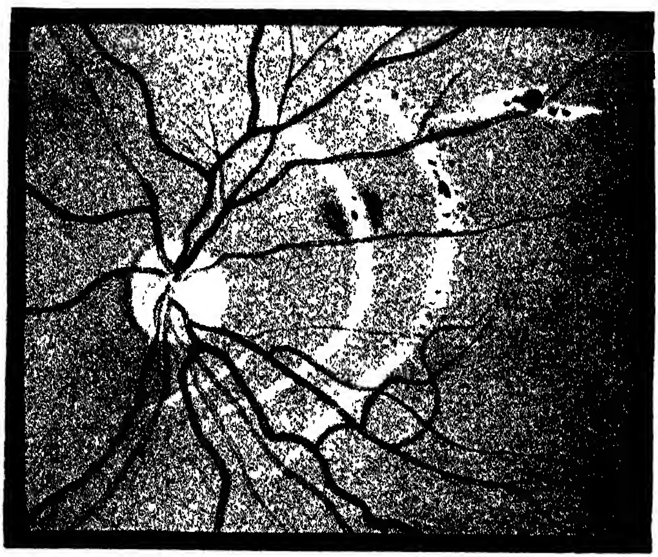


FIG. 2.—Ruptured choroid.

tension of irido-cyclitis can be explained by defective filtration of the aqueous at the angle of the anterior chamber owing to the viscous nature of the fluid, though capillary dilatation may play a part (*vide* p. 17). Secondary glaucoma is almost invariably associated with mechanical obstruction at the same spot. Usually the obstruction takes the form of adhesion of the iris to the back of the corneo-sclera. This peripheral anterior synechia causes the obliteration of the network of the ligamentum pectinatum iridis, and prevents the fluid from gaining access to the canal of Schlemm. It is therefore imprisoned within the eye and the intraocular pressure rises. In order that this may occur it is necessary that the angle should be obliterated over a considerable part of its circumference, but there is a great tendency in most cases for the cause which has produced partial peripheral anterior synechia eventually to complete the circuit. In some cases in which secondary glaucoma supervenes there is no actual synechia, but the meshes of the ligamentum pectinatum are choked with leucocytes, pigment granules, fibrin, &c., so that filtration is inefficient.

Though peripheral anterior synechia seems definitely to be the immediate cause of secondary glaucoma, it is itself produced by a variety of conditions, most of which are various forms of anterior or posterior synechia.

When an anterior synechia is formed the plane of the iris is advanced, so that the angle of the anterior chamber on this side is made more acute than normal. The cause which led to the formation of the synechia also causes iritis, usually of the traumatic type. Traumatic iritis is merely a plastic iritis due to injury. In it much exudation is poured out, possessing great tendency to organise. It collects in the diminished angle and becomes transformed into fibrous tissue, which welds the iris and corneo-sclera together, thus producing a peripheral anterior synechia, which may be strictly localised, so that no secondary glaucoma supervenes. Such eyes, however, are liable to fresh attacks of iritis, often of an insidious character. Each attack is followed by further occlusion of the angle, until finally the amount remaining open is insufficient to carry out efficient filtration and the pressure rises.

The chief causes of secondary glaucoma are the following:

(1) *Perforation of the cornea with anterior synechia.* The perforation may be due to an ordinary perforating wound of the cornea with incarceration of iris in the scar, or it may be due to a perforating corneal ulcer. The wound may be due to

an operation, *e.g.*, extraction of cataract, for a peripheral section through the corneo-scleral margin or actually in the sclerotic near the margin has a similar effect to a wound in the cornea. The synechia need not necessarily be of iris, but after cataract extraction is often one of the lens capsule, which has the same effect of advancing the contiguous parts of the iris and obliterating the angle. Secondary glaucoma after operations may also be due to other causes (*vide* p. 505).

(2) *Annular posterior synechia* (*vide* p. 260). This acts by interposing an impermeable barrier between the posterior and anterior chambers. The lymph secreted by the ciliary body is thus prevented from passing forwards into the anterior chamber. The iris becomes bowed forwards—bombé—and the periphery becomes apposed to the corneo-sclera, where it later becomes adherent. The aqueous is thus unable to escape from the eye and the tension rises. If the condition is not relieved by operation secondary glaucoma causes blindness. The prolonged high tension then causes degeneration of the ciliary processes, which cease to produce so much fluid, so that finally the tension may be normal or even sub-normal, and the eye may shrink. The condition is relieved by iridectomy, or if this is impossible, by iridotomy, communication between the posterior and anterior chambers being thus restored.

(3) *Wounds of the lens.* When the lens is wounded it swells (*vide* p. 450), and pushes the iris forwards into contact with the corneo-sclera. Moreover the swollen lens matter in the aqueous impedes filtration through such part of the angle of the anterior chamber as remains open, both mechanically and also by increasing the albuminous content of the aqueous. Mere apposition is sufficient to produce permanent secondary glaucoma, which should at once be relieved by operation (*vide* p. 450). If it is not performed the iris becomes adherent to the corneo-sclera and the glaucoma becomes permanent, although the lens eventually may be absorbed.

(4) *Dislocation of the lens.* This may be complete through the pupil into the anterior chamber. It then blocks the angle, especially if the iris is firmly contracted against its posterior surface. Partial lateral dislocation of the lens causes it to push forwards the iris on the side towards which it is dislocated. Since the circle of the equator of the lens is not much smaller than that of the angle a considerable portion of the latter may be blocked, and secondary glaucoma supervenes.

(5) *Intraocular tumour.* The mechanism whereby this produces secondary glaucoma will be described later (*vide* p. 425).

(6) *Intraocular hæmorrhage.* Severe intra-vitreous or sub- (a) choroidal hæmorrhage forces forwards the vitreous and lens so that the iris is pushed into contact with the cornea. It also acts by filling the eye with highly albuminous fluid which filters with difficulty. If the vessel which has ruptured is large the tension may be raised to that of blood pressure.

A special type of glaucoma is sometimes met with after retinal hæmorrhage, which may be due to some unknown cause (b) or to thrombosis of the central vein (*q.v.*). It is probably caused (c) by mixture of the lymph with albuminous fluids. Such cases

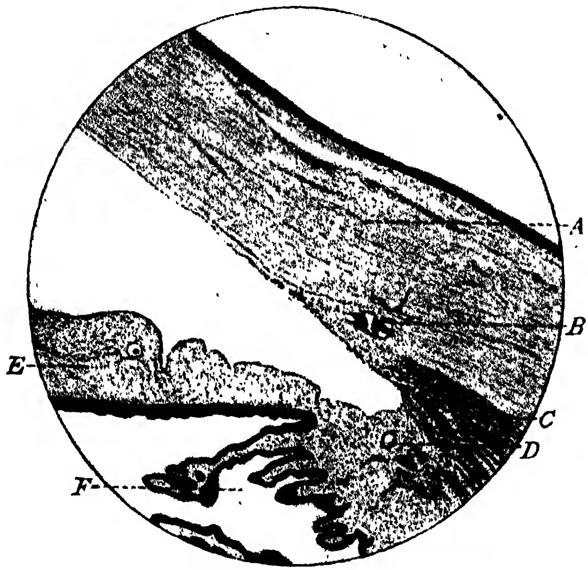


FIG. 160.—Normal angle of anterior chamber.

are sometimes grouped together under the designation *hæmorrhagic glaucoma*, a term which is however best avoided. They may be indistinguishable from primary glaucoma if not seen until the media are too opaque for ophthalmoscopic examination. Iridectomy is likely to be accompanied by severe hæmorrhage, and is therefore contra-indicated. Imp

Primary Glaucoma. The cause of primary glaucoma is unknown, but several factors are recognised as having a predisposing influence. These factors may be considered under two heads—the type of person affected and the type of eye involved. With regard to the type of person, primary

glaucoma is essentially a disease of late adult or advanced life. There is a certain racial tendency and it is common in Jews. It is sometimes hereditary, and in these cases affords examples of "anticipation" (Nettleship), *i.e.*, it occurs at an earlier age in each succeeding generation. It is commoner in women, who are more liable to venous congestion in various parts of the body. The acute manifestations of the disease are prone to occur in those who show an instability in their vaso-motor reactions and a weakness of a normal balance

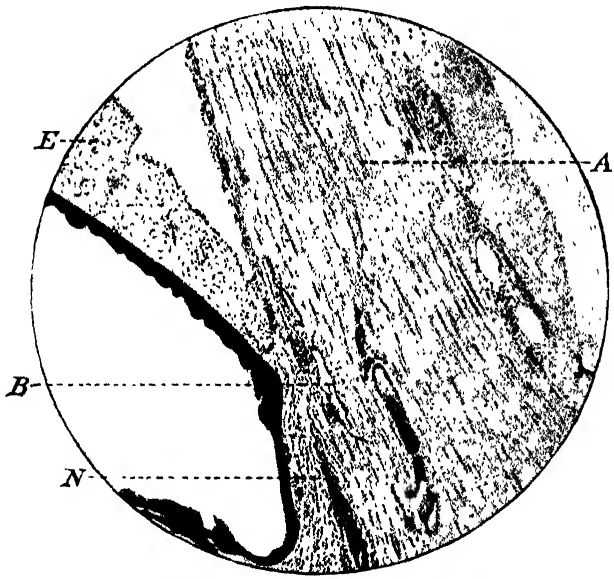


Fig. 161.—Peripheral anterior synechia (*N*), causing blockage of the filtration angle. *A*, cornea; *B*, canal of Schlemm; *E*, iris.

between the sympathetic and parasympathetic systems. Strain. overwork, anxiety and nervous shock are definite ætiological factors in this type of case. It would seem that a lack of the normal vasomotor control of the capillary circulation of the eye is an essential factor in many cases. On the other hand more chronic cases are frequently associated with arterio-sclerosis and vascular degeneration. There is, however, no significant correlation between a high blood pressure and the incidence of glaucoma for it is to be remembered that a high systemic pressure is often associated with a low capillary pressure.

The type of eye is also of significance. Hypermetropic eyes are more susceptible than those with normal or myopic refraction, due to the fact that a hypermetropic eye, although not necessarily so, is usually small. In such an eye the size of the lens is of considerable importance. Priestley Smith originally pointed out that since the lens continues to grow throughout life (*vide* p. 9) the space between the equator of the lens and the ciliary processes, the circumlental space, will become smaller as the patient becomes older. Since the size of the lens varies a little with that of the eye, if the eye is small this space may become so diminished that slight congestion of the ciliary processes may bring them in contact with the lens. The effect will be to prevent the fluid formed by the ciliary body from passing forwards through the pupil. The lens will therefore be forced forwards, and will push the iris in front of it, making the anterior chamber very shallow, and bringing the periphery of the iris in contact with the corneo-sclera. In this manner the filtration angle will be occluded and glaucoma will supervene.

It has been found that the size of the cornea is a good criterion of the size of the ring formed by the ciliary processes, *i.e.*, of the circumlental space.

A further point is that as age advances the anterior chamber becomes shallower. This will have the effect of diminishing the size of the angle of the anterior chamber, and still more so if the cornea is small. Filtration is carried out less easily when the meshes of the ligamentum pectinatum iridis are crowded together than when they are widely separated. Moreover the fibres of the ligamentum pectinatum tend to become thickened and sclerosed in elderly people. In these eyes a very slight further diminution of the angle may bring on an attack of glaucoma. Thus, the mere dilatation of the pupil with a mydriatic, by folding up the iris so that it is crowded into the angle, may suffice to occlude it entirely; *hence the extreme danger of instilling a mydriatic into the eyes of elderly people*, especially if they are hypermetropic or have small corneæ and shallow anterior chambers.

Since both eyes usually have a similar structure, glaucoma is likely to be bilateral, but one eye is generally affected before the other.

In summary, therefore, it may be said that glaucoma tends to occur in elderly persons who show either vascular instability or sclerosis; and the eyes affected are usually

hypermetropic with a small cornea, a (relatively) large lens with sclerosis of the pectinate ligament.

The anatomical effects of pathological increased intraocular pressure are as follows. The already shallow anterior chamber is made still more shallow. In an early acute attack the periphery of the iris is merely apposed to the corneo-sclera. In the later stages and in chronic glaucoma of some standing it is firmly adherent. The longer the condition has lasted the firmer is the union. The iris is first bound down by organised exudate; later, the iris stroma atrophies and the inner wall of Schlemm's canal, which may be almost obliterated, is covered only by degenerated retinal pigment epithelium. Anterior to this a "false" angle is formed where the iris leaves the cornea; no filtration can take place either through the peripheral anterior synechia or through the false angle.

The part of the eye which suffers earliest and most from the increased pressure is the head of the optic nerve. The lamina cribrosa, which is more resistant than the nerve tissue, is less resistant than the sclerotic. Hence it becomes pushed backwards, the nerve fibres being depressed also. The first manifestation of the effects of pressure is a bowing backwards of the connective tissue which forms the lamina cribrosa, so that it becomes concave anteriorly instead of passing straight across the porus opticus. This effect continually increases, until the lamina cribrosa is displaced backwards as a whole. Meanwhile the nerve fibres have been pressed together, so that the papilla becomes flat or depressed. The pressure causes the nerve fibres to atrophy, so that finally the lamina cribrosa is exposed upon the surface. In the final stage a deep cup is formed, generally having overhanging edges.

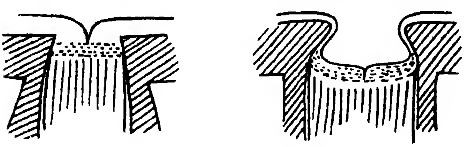


FIG. 162.—A, diagram of meridional section of normal disc; B, diagram of meridional section of glaucomatous cupped disc. Note the displacement backwards of the lamina cribrosa.

The steady or recurrent, often only moderately raised pressure of chronic glaucoma is more liable to cause cupping of the disc than the rapidly induced high pressure of acute glaucoma. Hence the disc may appear normal after the relief of acute glaucoma by operation. It must be remembered, however, that the acute attack may be superposed upon a long-standing chronic glaucoma.

Pulsation of the arteries at the edge of the disc is often seen in glaucoma. While venous pulsation is of little importance, spontaneous arterial pulsation is always pathological (*vide* p. 127). It is not always spontaneous in glaucoma if the tension is not very high, but even then it is induced by very slight pressure of the finger through the lid. The arterial pulsation is due to the increased pressure upon the walls of the vessels, so that the intravascular pressure is only able to force blood through at the height of the cardiac systole.

Other parts of the eye show less change. The pressure causes degeneration of the nerve fibre layer of the retina. The choroid becomes degenerated and thinned, only the larger vessels remaining. The ciliary body becomes degenerated in the last stages, after which the tension may cease to be raised owing to defective secretion of lymph.

The subjective effects of pathological increased intraocular pressure are manifold. Pain is complained of, due to stretching of the sensory nerves of the eye. The patient sees coloured haloes round lights; these are due to alteration in the refractive conditions of the corneal lamellæ. The colours are generally distributed as in the spectrum, with red at the outer margin in the ring. The pupil becomes slightly dilated and immobile, owing probably to œdema and pressure on the ciliary nerves as they run through the choroid. Rapid diminution in the amplitude of accommodation may be a prominent feature, so that there is an apparent increase in presbyopia. It is attributable to pressure on the ciliary nerves and on the ciliary muscle. Diminution of vision is due to cloudiness of the media, retardation of the blood flow, and pressure on the nerve fibres in the retina and optic papilla. Cloudiness of the

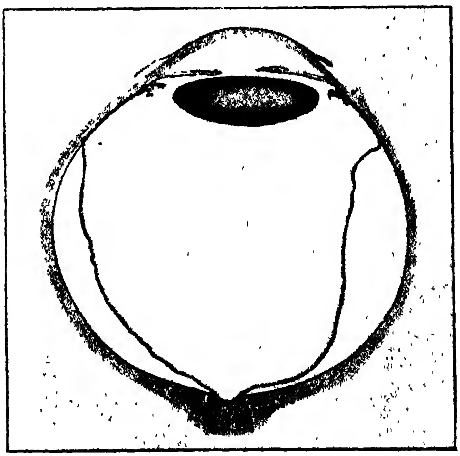


Fig. 163.—Horizontal meridional section of a glaucomatous eye.

media affects the cornea principally, and is due in the early stages to altered refractive conditions, in the later to œdema. Pressure on the nerve fibres first affects the temporal side of the retina, and therefore the nasal side of the field. Later, the field becomes contracted in all directions, and central vision is depressed. Finally, vision is abolished, owing to total atrophy of all the fibres.

Acute Glaucoma. In even the most acute cases of glaucoma careful inquiry will often elicit prodromal symptoms. Transient attacks of obscuration of vision, as if a cloud were in front of the eye, have occurred. Bright lights have appeared to be surrounded by rainbow haloes. A feeling of discomfort in the eye and neuralgic headache accompany these symptoms. Such attacks become more frequent, come on especially at night and after excitement or worry. It is noticed that stronger glasses are required for near work. This condition may extend over months or years.

If the patient is examined while the symptoms are present it will be found that there is a slight haziness of the cornea, so that it looks bluish, like glass that has been breathed upon—hence the term glaucoma (γλαυκός, sea green). If the field of vision is taken between the attacks some slight contraction of the nasal field may be found, but central vision may be perfect.

The acute attack sets in suddenly. It may be due to some condition inducing venous congestion, *e.g.*, constipation, menstruation, over-feeding, alcohol, &c., or to worry, fatigue, a recent illness, or to the instillation of a mydriatic. The pain and anxiety of acute glaucoma in one eye may induce an attack in the other. Intense pain is felt in the eye and over the distribution of the fifth nerve. The pain is frequently so bad that it causes vomiting, and the attack is liable to be mistaken for a severe "bilious attack." The temperature may be raised. The constitutional disturbance is often so great that the patient is prostrated, the pulse becoming irregular and intermittent. The vision rapidly diminishes, so that in a few hours only hand movements can be recognised. In a considerable number of cases both eyes are affected almost simultaneously.

Objective examination shows some œdema of the lids and conjunctiva ; the latter is intensely congested and looks dusky red, owing to the dilatation of the veins. Ciliary congestion is marked. The cornea is cloudy and insensitive to the touch. The anterior chamber is very shallow. The iris is discoloured, the pupil moderately dilated and oval, generally with the long axis vertical. The reactions to light and accommodation are

abolished. Ophthalmoscopic examination is impossible owing to the cloudiness of the cornea. The tension of the eye is considerably raised.

There is no true inflammation in the early stages, so that the term inflammatory glaucoma, frequently used, is inadvisable; it should be replaced by *congestive glaucoma.* The condition is probably due to a widespread capillary dilatation in the uveal tract, caused in part, perhaps, by the liberation of histamine in the tissues. This view is supported by the proved presence of histamine in the aqueous and vitrious in the glaucoma of epidemic dropsy in India (Kirwan). This would account for the failure of adrenaline and its compounds (glaucosan, lævo-glaucosan) to relieve the congestion (*vide* p. 17); it would also account for the failure of choline derivatives, *e.g.*, doryl, and histamine in the form of amino-glaucosan (*vide* p. 64).

If the condition is not relieved by operation, the amount of permanent diminution of vision depends upon the severity and duration of the acute attack. Total abolition of vision may result. More frequently improvement occurs, ushered in by diminution of pain. Considerable lowering of the visual acuity, and, still more, contraction of the field, follows every acute attack. All grades, indeed, may be met with, from the mild prodromal attacks to the severest, with complete blindness. The tension remains permanently slightly elevated. Some congestion and irritability persist. The pupil reacts sluggishly, and the iris shows signs of atrophy, usually first in one or more sectors. Ophthalmoscopic examination now becomes possible. Cupping of the optic disc may or may not be found, according to the duration of the raised tension before and after the acute attack. A single acute attack is not followed by cupping immediately, for this demands more or less prolonged high tension.

In every disease of one eye the other should be thoroughly examined. In acute glaucoma it may be found that chronic glaucoma has existed long unobserved in the other eye, and well-marked cupping of the disc may be present. The same causes which induced the acute attack in one eye may rapidly induce a similar attack in the other. The pain and worry associated with preparations for operation and so on increase the danger. This eye should therefore be carefully watched, and prophylactic measures adopted. It is usually sufficient to instil a drop of 0·5 per cent. solution of eserine in the sound eye every day so as to keep the pupil contracted.

It is of the utmost importance that pathological cupping

of the disc should always be recognised when present (Plate VIII., Fig. 2). When fully developed it differs in ophthalmoscopic appearance from a deep physiological cup, with which it is most likely to be confounded, in that the excavation reaches to the edges of the disc and the sides are steep, not shelving. The retinal vessels have the appearance of being broken off at the margin of the disc. If they are accurately focussed here their continuations upon the floor of the cup are slightly out of focus and look broader and paler. When the edges overhang, as is often the case, the course of the vessels as they climb the sides of the cup is hidden. By the indirect method slight lateral movement of the large lens causes a distinct parallax (*vide* p. 117), which is more marked the deeper the cup. By the direct method the difference in level between the vessels at the edge and on the floor can be measured (*vide* p. 121).

There is always some atrophy of the optic nerve when the disc is cupped by the glaucomatous process; it is therefore not surprising that there may be great difficulty in distinguishing a shallow glaucoma cup from the slight depression which follows simple atrophy of the nerve without increase of tension (*vide* p. 401). If the cup is deep and total it is certain to be glaucomatous, except in the rare cases of ectatic coloboma of the disc (*q.v.*). In shallow glaucomatous cups the disc has a pink colour, whereas the atrophic cup is white. In many early cases all the conditions have to be weighed carefully before it is possible to come to a definite conclusion; the field of vision usually affords the most important criterion, the contraction being chiefly nasal in early glaucoma, concentric in optic atrophy.

The final stage of the untreated disease is *absolute glaucoma*. The eye is completely blind. The anterior ciliary veins are dilated, and a reddish blue zone surrounds the cornea. The cornea is clear, but insensitive; it may have vesicles (bullous keratitis) (Fig. 148) or filaments (filamentary keratitis) upon it. The anterior chamber is very shallow. The iris is dilated, atrophic, and may have a broad zone of pigment around the pupil (ectropion of the uveal pigment). The pupil is grey or greenish, instead of jet black. The optic disc is deeply cupped. The tension is high; usually the

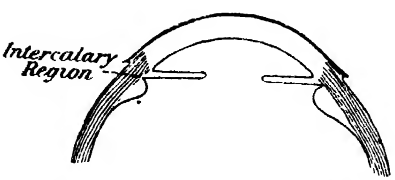


Fig. 164.—Diagram showing intercalary region.

eyeball is as hard as stone. Such an eye is generally painful, with temporary exacerbations, though patients often prefer to bear the pain rather than submit to excision. If it is still retained degenerative changes occur. The more important are due to giving way of the sclerotic before the continued high intraocular pressure. In this manner scleral staphylomata are produced. They may be in the neighbourhood of the ciliary body—ciliary staphylomata, or at the equator—equatorial staphylomata.

Anatomical investigation shows that *ciliary staphylomata* are of two kinds. In one, the region where the iris is adherent to the corneo-sclera gives way (Fig. 164). These are called *intercalary staphylomata* (Fig. 165). In them the iris projects into the anterior chamber from an attachment at the anterior

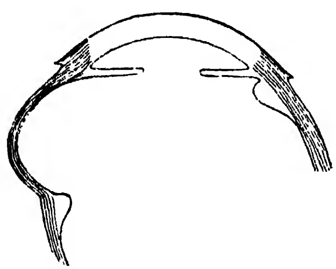


FIG. 165.—Diagram showing intercalary staphyloma.

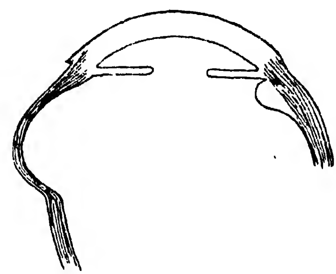


FIG. 166.—Diagram showing true ciliary staphyloma.

margin of the staphyloma, while the ciliary body, little altered, forms the posterior margin. The other form is the true *ciliary staphyloma* (Fig. 166). In this the region of the ciliary body itself gives way, so that it becomes spread out over the inner surface of the ectasia. In many cases both parts become ectatic. Clinically it is impossible to distinguish between these forms.

Equatorial staphylomata can only be seen clinically when the eye is turned well to one side and the lids separated. The thinning and bulging of the sclerotic occurs principally at the spots which are weakened by the perforation of the vortex veins and are unsupported by the recti muscles. Such globes may become enormous, with walls as thin as paper. There is considerable danger of rupture from slight injury.

Sooner or later the tension becomes normal or diminished in eyes with absolute glaucoma. This may be due either to

stretching of the walls as already explained, or to degeneration of the ciliary body, whereby its secretory functions are diminished or abolished. Usually both factors play a part, varying according to the particular case. Such an eye may even shrink, but more commonly ulceration of the cornea occurs, owing to the defective resistance of the degenerated tissues. Hypopyon ulcer, panophthalmitis, phthisis bulbi then form the sequence of events.

Diagnosis. Acute glaucoma is more likely to be mistaken for iritis than any other disease. The differential diagnosis has already been discussed (p. 261).

Treatment. Acute glaucoma demands immediate energetic treatment. It is imperative that the tension shall be reduced as soon as possible. Theoretically this is best and most permanently effected by immediate operation, as in many cases other measures fail. The moment is, however, an unfavourable one for operation. The conjunctiva is chemosed, the anterior chamber is extremely shallow, there is no time for exhaustive preparations, and general or retrobulbar anæsthesia will be necessary owing to the impermeability of the stretched cornea to local anæsthesia. Owing to the constitutional disturbance and the irregular action of the heart, one may feel diffident about giving a general anæsthetic in these cases. The danger is liable to be over-estimated.

It is permissible to try other remedies for a short time first. It is first essential to draw the congested iris away from the filtration angle. Miotics, in addition to this result, relieve vascular congestion and œdema by their vasodilatatory effects. Eserine (1 per cent.) is instilled into the affected eye at five minutes' intervals for half an hour, and then hourly, until the intraocular pressure is reduced or an operation becomes imperative. Eserine (½ per cent.) should be instilled into the other eye and continued twice daily during the critical state of the affected eye. The action of eserine is assisted by medical diathermy. An eye pad, composed of layers of cotton wool wrung out in warm saline and applied evenly to the closed lids, is attached through a special headband to one of the electrodes, the other being bound to the arm. The current is slowly increased until the heat is as strong as the patient can bear. This is generally between 300 and 600 milliamperes : it is maintained at this reading for five minutes and then slowly reduced to zero. An injection of morphia (gr. ¼) also helps to relieve pain, and acts as a miotic.

Rectal administration of 6 ounces of 50 per cent. magnesium sulphate is more efficacious in reducing the intraocular pressure than the intravenous injection of hypertonic saline (50 c.c. of 30 per cent. sodium chloride), which is not free from risk, especially in patients with albuminuria.

The application of leeches to the temple assists the reduction of congestion. Hot bathings are given hourly in the intervals of diathermy, which is not used more than twice a day.

Even if the results are satisfactory trephining must be performed when the eye is quiet. If it is imperative to operate in the acute stage, iridectomy should be performed, not trephining. There is, indeed, one objection to palliative and non-operative treatment, even when it is successful, viz., that the patient may refuse the radical operation when the acute stage has passed off. In cases in which this is to be feared, it is advisable in the patient's interest to perform the operation at once. On the other hand the ultimate result is more satisfactory if trephining can be done—for one reason because it causes less astigmatism than iridectomy. If there should be a lacrymal mucocele (*vide* p. 653) present both canaliculi should be ligatured, or the puncta cauterised with the actual cautery. If both eyes are affected, both should be operated upon at the same sitting. In no other disease is this procedure indicated or even justified.

If the intraocular pressure remains high in spite of treatment an immediate operation is necessary. The classical operation is iridectomy (*vide* p. 476), and the results have usually been very good. It has the disadvantage of causing some astigmatism, which is avoided by trephining; but owing to the extreme congestion of the eye the technical difficulties of the latter operation are increased. These, however, have been much reduced with modern methods of anæsthesia by intravenous sodium pentothal, which lowers the blood pressure and incidentally the intraocular pressure also. It is possible to prevent a sudden reduction of the intraocular pressure by withdrawing the trephine very slowly. A wide peripheral iridectomy should be done, leaving the sphincter pupillæ intact.

Iridectomy was first tried for glaucoma by von Graefe (1856) on the erroneous theory that as it lowered the tension of the normal eye (which is not true) it would be beneficial in glaucoma. The true explanation of the efficacy of iridectomy in glaucoma is that it reopens the angle of the anterior

chamber and thus restores filtration. In acute glaucoma the iris is at first merely apposed to the corneo-sclera at the periphery. The manipulations draw it away. Part of the iris is then torn away at its ciliary attachment, and the angle of the anterior chamber is reopened in this situation. There is some reason to think that fluid is absorbed by the cut edges of the coloboma, for wounds of the iris do not heal by cicatrisation, the lymph-spaces of the stroma always remaining continuous with the anterior chamber at the cut margins.

In absolute glaucoma pain is best relieved by hot bathing and internal administration of aspirin. If possible consent

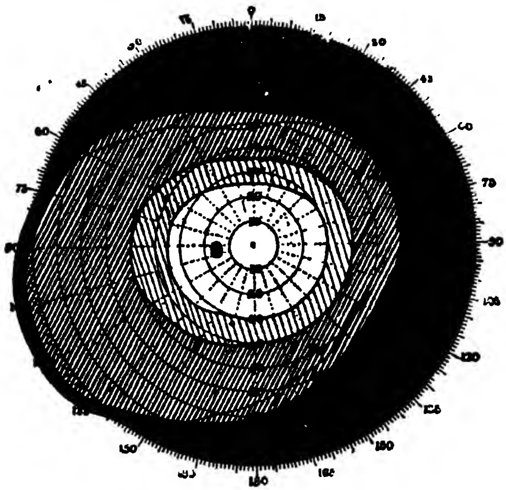


FIG. 167.—Normal fields with tests as follows:—10/300, 6/2000, 3/2000 (*vide* p. 141). (A. H. H. Sinclair.)

should be obtained to excise the eye. If this is refused the pain may be relieved for a time, varying in different cases, by a retro-ocular injection of 1.5 c.c. of novocain (4 per cent.), followed seven minutes later by alcohol (80 per cent.). A firm pad and bandage is applied for twenty-four hours. If the pain recurs this treatment can be repeated. It is rarely justifiable to trephine or perform any other operation merely for the relief of tension in these eyes, since there is nearly always a risk that the cause of the glaucoma may be an intra-ocular malignant growth, usually sarcoma of the choroid.

Chronic Glaucoma, sometimes called simple glaucoma, is fundamentally the same disease as acute primary glaucoma;

every grade of severity is met with, but the more chronic forms are so insidious that special attention must be directed towards their discovery.

The patient usually complains of transient attacks of obscuration of sight, and of gradually diminishing acuity of vision; but in some cases there may be no history of haloes and the diminution of vision is continuous and very insidious.

The eyes may appear perfectly normal at the first examination, though sometimes the anterior ciliary veins are congested, and the pupil is somewhat dilated and sluggish. An abnormally small cornea should draw attention to the possibility of glaucoma, and hypermetropia increases the probability. The

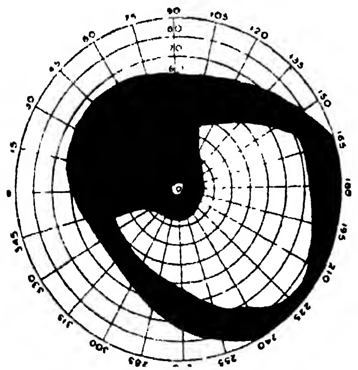


FIG. 168.—Field of vision in commencing glaucoma, showing double arcuate scotoma and quadrantic defect (5/330).

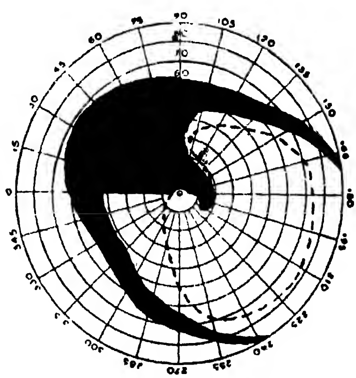


FIG. 169. – Glaucoma fields with sector defect and arcuate scotoma (5, 2/330).

tension may be quite normal, and is found to be elevated only during an attack of cloudy vision. Hence it may be necessary to examine the patient frequently and at various times in the day, especially during such attacks.

Subjective examination will often reveal no diminution of central vision. Hence it is of the utmost importance to take a careful chart of the field of vision. The commonest change is partial loss of the nasal field, often accompanied by some general contraction (Fig. 168). The change in the nasal field may be regular in outline, or there may be indentations with the apex directed towards the fixation spot. Such sectorial defects may be above or below. In later stages the general contraction is more marked, and eventually only a paracentral patch of the

temporal field persists, central vision being abolished. Partial scotomata are to be found by special means before nasal constriction occurs, or when it is only slightly developed. If the central area of the field is mapped out on a Bjerrum's screen (*vide* p. 143), or with a suitable scotometer, an area of relative defect can frequently be traced in direct continuity with the blind spot (*Bjerrum's scotoma*). The scotoma may pass in an arc from the blind spot above or below the fixation point, or may form a complete annular scotoma. It is due to injury of bundles of nerve fibres at or near the edge of the disc. The destruction of these fibres is also said to account for a characteristic sharply defined horizontal edge to the lost portion of the field on the nasal side (*Rönne's step*) (Figs. 168, 169). The earliest sign of all is said to be a sickle-shaped extension of the blind spot above or below, or both, with the concavity of the sickle directed towards the fixation point (*Seidel's sign*); this is of more doubtful significance. Paracentral scotoma may persist with a full peripheral field for months or even years, but eventually the characteristic constriction of the nasal field sets in, and then often progresses rapidly. A relative central scotoma sometimes follows rapidly on the development of the paracentral scotomata: the prognosis is worse in these cases.

Defective light sense is probably always an early feature of chronic glaucoma. The light-minimum is raised and dark adaptation is slowed, so that patients take longer to get used to the lower degree of illumination in passing into a dimly lighted room, a disability which becomes increasingly disturbing in the later stages.

Ophthalmoscopic examination will often show some cupping of the disc; frequently it is far advanced, though the symptoms have been so slight as to have passed almost unnoticed. The field in nearly all these cases will be found to be much damaged.

Primary glaucoma invariably attacks both eyes sooner or later; usually one is considerably more advanced than the other. The chronic form sometimes occurs in young people, and seems to attack men almost as frequently as women. It also occasionally occurs in myopic eyes.

There is no evidence that chronic glaucoma is due to increased blood pressure, though, so frequently occurring in elderly patients, it is often associated with it. Although the intraocular pressure responds passively to rapid changes in the general blood pressure (*vide* p. 19), slow changes are com-

pensated and a fresh equilibrium of secretion and excretion is established.

Diagnosis. Chronic glaucoma has frequently been mistaken

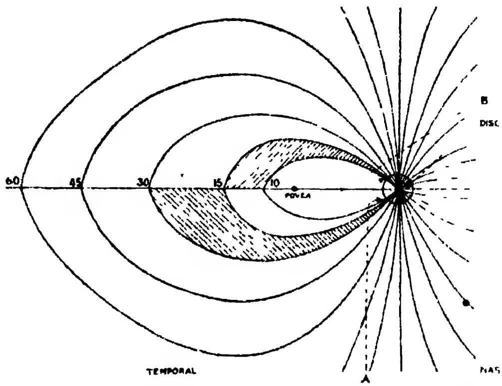


Fig. 170.—The course of the nerve fibres in the retina: showing fibres involved by lesions at A and B.

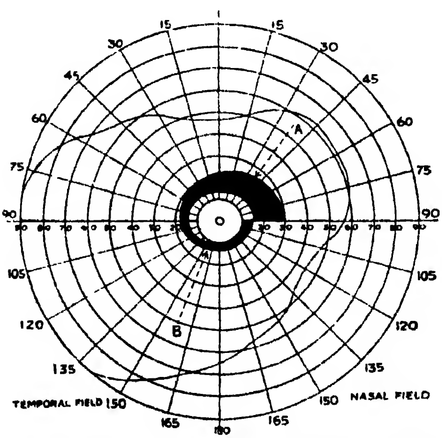


Fig. 171.—Arcuate scotomata in field of vision corresponding to lesions A and B.

for cataract or optic atrophy. Cases occur in which the gradual loss of vision is attributed to cataract, and the patient is told that nothing can be done until the cataract is ripe for operation. Vision is thus irretrievably lost. As a rule diagnosis is very easy. The haze of the pupil is bluish, diffuse and uniform.

unlike the usual appearance in cataract. The pupil may be slightly dilated and generally reacts less to the stimulation of light. Examination with the ophthalmoscopic mirror in most cases renders the diagnosis certain by showing a uniform red reflex and the absence of opacities in the lens.

Doubt as to the presence of glaucoma may arise in cases with senile striæ in the lens. In patients predisposed to glaucoma the swelling of the lens which occurs in the early stages of cataract (*vide infra*) may lead to increase of the intra-ocular tension. The disc is not usually cupped in these cases, but there is generally more failure of vision than is accounted for by the lenticular opacity, and the field of vision may show contraction on the nasal side; the reaction of the pupil to light may be sluggish. In such cases it is advisable to do a preliminary iridectomy, which should be of the glaucoma type with a large and peripheral coloboma.

Cupping of the disc in glaucoma is accompanied by atrophy of the nerve fibres, and it may be difficult to distinguish this atrophy from optic atrophy due to other causes, especially those giving rise to what is known as "primary" optic atrophy. The latter condition shows some depression of the surface of the disc which is usually too slight to be actually measured by the ophthalmoscope, but is demonstrated by the bending of the vessels as they pass over the edge of the disc. The depression is greater in most cases of chronic glaucoma. In cases which give rise to difficulty in diagnosis little aid is afforded by the tension of the eye, which may be normal or inappreciably raised at the time of examination. Reliance must be placed upon the history of the case, the condition of the cornea and anterior chamber, and the record of the field of vision. The latter shows concentric constriction in primary optic atrophy, more marked contraction of the nasal side in glaucoma. Accurate mapping out of the blind spot and the central region of the field on a large scale reveals changes in glaucoma (*vide supra*) which do not occur in optic atrophy.

Treatment. As soon as the diagnosis is made miotics, pilocarpine (½ per cent.) or eserine (¼ to ½ per cent.) twice a day, should be instilled. The eyes may be kept under observation for a time unless the disease is far advanced. If miotics fail to control the intraocular pressure or if the loss of the visual field continues to progress operation must be undertaken. *Miotics never cure chronic glaucoma*. Hence they must be adopted only as a temporary means of alleviation or as useful adjuncts. They cause some conjunctival and ciliary

congestion if used constantly. Pilocarpine is the less irritating, but perhaps less efficient. If these fail "doryl," a choline derivative, may be used in 0·75 per cent. solution. Gentle massage with the finger-tips is certainly useful, promoting lymph-flow, and temporarily reducing tension.

Until a few years ago iridectomy was the operation invariably performed for the relief of chronic glaucoma. The prognosis of iridectomy, however, is not nearly so good as in the acute form, owing to the fact that the periphery of the iris is often firmly adherent to the corneo-sclera before the condition is diagnosed. Hence a special endeavour was made to open up the occluded angle. If the section is made at the apparent corneo-scleral margin, when the iris is torn away it is almost certain to tear at the false angle, and little or no good results. It was customary, therefore, to make the section as peripheral as is consistent with safety to the ciliary body, *i.e.*, 2 mm. behind the corneo-scleral margin, with the object of carrying the incision through the adherent parts of the iris. It must be confessed that this object was seldom if ever attained.

It was found, however, that this method of performing iridectomy sometimes succeeded in spite of the impossibility of restoring the normal method of filtration. It effected this by establishing a *filtering scar*. The new scar in these cases is composed of spongy tissue, through the interstices of which the intraocular fluid is able to make its way into the subconjunctival tissue, where it is absorbed.

Deliberate attempts have therefore been made to establish a safe filtering scar for the relief of chronic glaucoma. Such a scar is usually formed only if there is some impediment to proper cicatrisation, as, for example, when the iris is incarcerated in the wound (*iridencleisis*). There is some danger in leaving a knuckle of iris in the wound when doing an iridectomy, since such a procedure involves grave risks of iridocyclitis, secondary infection of the eye, and sympathetic ophthalmia. Good results, however, have been obtained by Holth's method.

In an ordinary corneo-scleral section the lips of the wound are in good apposition and sound healing rapidly takes place. This is much less likely to occur if there is a gap between the lips of the wound. Under these conditions the gap becomes filled with loose scar tissue and a filtering cicatrix may result. Various operations have been based upon this principle. In *Lagrange's operation* an ordinary iridectomy is performed, but before closing the wound a small piece of the anterior lip is

snipped off without wounding the conjunctival flap. In *Herbert's operation* a small rectangular trap-door is cut in the sclerotic just outside the limbus, the hinge being towards the cornea. A circular wound in the sclerotic offers the best chance of success theoretically.

Trephining is the operation which is now generally performed for chronic glaucoma (*vide* p. 482). By it a disc 1·5 mm. in diameter is removed from the wall of the globe just inside the limbus, so that part lies in the cornea and part in the sclerotic. The knuckle of iris which prolapses into the wound should be excised, so that a small peripheral iridectomy is performed. If a larger disc is removed there is danger of the tension becoming permanently too low, with the risk of malnutrition of the eye. The operation sometimes fails from blockage of the wound with iris or too dense scar tissue. Filtration may be encouraged by gentle massage of the eyeball through the upper lid. Secondary infection of the eye is liable to occur from injury of the epithelium covering the bulging conjunctiva, but it is rare and is a risk which may be taken justifiably in treating so grave a disease as glaucoma. If trephining fails it can be repeated at some other part of the limbus, but the wound should always, if possible, be placed where it is covered and supported by the lid, though it should not be too close to the previous site.

Trephining is uncertain in its results and is liable to be complicated with serious dangers, both immediate and remote, yet in my opinion it affords the best means on the whole yet devised for dealing with chronic glaucoma. It seldom causes any improvement in vision or in the field of vision, but it usually prevents further loss.

In very advanced cases the field of vision may be found reduced almost to the fixation point. Experience teaches that in these cases operation may not only do no good, but may result in the sudden complete loss of all vision. After explaining this risk to the patient it is still advisable in most cases to perform the operation, because this unfortunate result is rare, and in any case the eye will become blind and probably painful unless the tension is relieved.

Since glaucoma always occurs in the other eye sooner or later the question of a prophylactic operation in this eye arises. Since it is attended by some danger, both immediate and remote, and since the advent of glaucoma may be long delayed, it is inadmissible to operate until some slight contraction of the nasal field can be demonstrated. The greatest care must

be taken to warn the patient of the danger of the disease attacking the other eye, and of the earliest symptoms. He should be examined thoroughly and the field of vision taken every three months, and he should be instructed to consult the surgeon at once if any signs of the disease occur. Weak pilocarpine or eserine drops may be used every other day as a prophylactic measure, and the general régime should be ordered so as to avoid cerebral congestion. He should also be warned against putting drops, lotions, or ointment of any kind into his eyes without the advice of an ophthalmic surgeon.

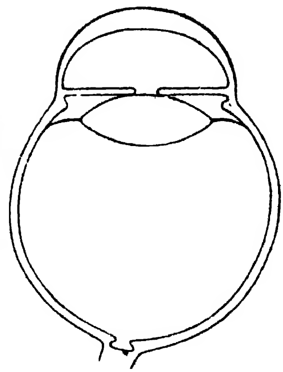


FIG. 172.—Diagram of eye with infantile glaucoma (buphthalmia). Note the stretching of the corneo-sclera at the periphery, the flattening and displacement backwards of the lens, the cupping of the disc, and the general enlargement of the globe.

When one eye has been almost or quite lost at the time of the first visit it becomes a serious question whether the better eye should not be operated upon rather than the worse. Many such difficulties arise in the treatment of glaucoma, and can only be decided by the conditions of the individual case.

Chronic glaucoma may arise in an eye with incipient cataract. Although trephining introduces a serious complication to subsequent extraction of the lens (*vide* p. 316) it should be performed in the usual way and in the usual situation. When the extraction is done later the upper part of the section should be in the cornea, slightly anterior to the trephine hole.

Cyclodialysis may be used to reduce the intraocular pressure in chronic glaucoma. It has been most successful in aphakic eyes; it should not be used in congestive cases. By it a channel is opened up between the anterior chamber and the suprachoroidal space. An incision about 3 mm. long is made in the sclera 4 mm. behind and concentric with the corneo-scleral junction in the lower temporal quadrant. A spatula specially curved to fit the inner aspect of the sclera is inserted and passed forwards between the scleral spur and the ciliary body into the filtration angle. Here it is swept transversely through a small arc, breaking down the ligamentum pectinatum and adhesions between the root

of the iris and the cornea. A conjunctival flap is sutured over the wound.

Infantile Glaucoma (*Syns.—Buphthalmia, Hydrophthalmia*). Glaucoma in rare cases attacks children, when it assumes a quite different clinical appearance. It arises from congenital blockage of the angle of the anterior chamber, due either to a congenital defect whereby the root of the iris does not become normally separated from the corneo-sclera or becomes adherent to it through intra-uterine or infantile inflammation. In many cases examined microscopically Schlemm's canal has been found defective or absent. The fundamental condition is therefore the same as in glaucoma in adults, viz., defective filtration of lymph from the eye. The reason why it assumes so different a clinical picture is dependent upon the greater plasticity and extensibility of the walls of the young eye. Instead of offering an insuperable resistance to increased internal pressure the sclerotic gives way more or less uniformly, so that the globe becomes very large.

The thinned sclerotic of the ciliary region is bluish in colour, owing to the uveal pigment showing through. The junction of the cornea and sclerotic also stretches, so that the cornea is forced forwards and assumes a globular shape (keratoglobus). The anterior chamber is therefore extremely deep (Fig. 172). There are often slight opacities in the cornea, some appearing as lines with double contour; these are due to ruptures in Descemet's membrane. In a considerable number of cases signs of keratitis develop accompanied by photophobia, and circum-corneal congestion associated with extensive opacities in the substantia propria, interfering considerably with the vision and precluding ophthalmoscopic examination. When first seen these cases may resemble an inflammatory condition. The lens does not participate in the general enlargement; owing to the expansion of the ciliary region the suspensory ligament is stretched so that the lens is flattened and displaced slightly backwards. This removes some support to the iris, which becomes tremulous (iridodonesis). The optic disc is deeply cupped if the condition has lasted long.

The intraocular tension is raised, but often scarcely appreciably as determined by clinical methods, owing to the expansion of the globe. This fact long prevented the true pathology of the disease from being recognised.

As a result of the expansion the eyes are usually myopic, though much less than might be anticipated from their length.

This is due to the flattening of the lens and its displacement backwards, as well as to some flattening of the cornea, all of which factors tend to counteract the axial myopia. There is usually astigmatism against the rule, owing to pressure by the lids on the plastic globe.

Both eyes are generally affected, and buphthalmia occurs in boys more often than in girls. Equilibrium may be established with no further loss of vision, but in other cases rapid deterioration occurs after puberty, perhaps due to increased rigidity of the walls.

Buphthalmia occurs frequently associated with neurofibromatosis (*vide* p. 647), and also with capillary nævus of the face and angiomatous conditions of the choroid and brain (*vide* p. 365).

Buphthalmia is to be distinguished from keratoglobus (*q.v.*).

Treatment is unsatisfactory. Miotics are useless so that operative treatment has to be attempted. Anterior sclerotomy with the trephine has frequently been the method of choice, but it usually has to be repeated several times and the visual results are often bad. Iridencleisis has occasionally given good results as has also irido-dialysis. More recently most success has followed the operation of trabeculotomy (goniotomy) introduced by Otto Barkan. In this operation a specially constructed knife introduced at the limbus is swept round the angle of the anterior chamber in the opposite segment of the eye. It is presumably effective by opening up the draining passages in those cases wherein the condition is due to the blockage of the corneo-iridic angle by persistent embryonic tissue.

CHAPTER XV

The Lens

The lens is composed entirely of epithelium, which is surrounded by a cuticular envelope or capsule (vide p. 9).

It is therefore subject only to metabolic changes, and is incapable of becoming inflamed. Degenerative changes in the lens invariably result in loss of transparency in the parts affected. This condition of partial or complete opacification is called *cataract*.

The most important chemical constituents of the lens are the salts and proteins. Spectroscopic examination reveals many metals, of which the most important are sodium, potassium and calcium. The potassium content diminishes with age, but the calcium is relatively constant (Adams), except that it is definitely excessive in cataractous lenses. The proteins consist of euglobulin, and α-, β-, and γ-crystallins. β-crystallin is soluble in water and decreases with age, a factor in the sclerosis of the nucleus. The crystallins are rich in tyrosine, cysteine, and leucine, amino-acids which tend to form melanins on exposure to ultra-violet light, thus accounting for the normal and pathological (*vide* p. 305) pigmentation of the lens. Owing to the absence of a blood supply the lens is dependent for its metabolism on an autoxidation system. This is a reversible oxidation-reduction reaction carried out by glutathione (Adams), a cysteine-like substance containing an SH group, which on oxidation changes into an SS group. The lens also contains a relatively large amount of vitamin C, which also probably acts with glutathione as a reducing agent, the reversible reaction being

Sugar + oxidized glutathione $\longleftrightarrow$ ascorbic acid + reduced glutathione

$$C_6H_{12}O_6 + G.SS.G \longleftrightarrow C_6H_8O_6 + 2G.SH$$

Thermostable substances (β-crystallin) in the lens effect reduction of this product (SH $\leftrightarrows$ SS). The mature cataractous lens contains no glutathione or vitamin C.

The earliest stage in the development of cataract is an accumulation of fluid either as droplets beneath the capsule or in spindle-shaped spaces between the lens fibres. Clinically this stage can be recognised by inequalities in the refractive indices of the fibres and fluid which give rise to light and dark

streaks when light is thrown into the eye by the mirror. The spots and streaks differ from the definite opacities which follow by the fact that if the mirror is tilted slightly the dark streaks become light and *vice versâ*. In the next stage coagulation of the proteins occurs, forming globular masses called Morgagnian globules. At a later stage the fibres break down into rounded masses which are indistinguishable from Morgagnian globules. These masses are definitely opaque.

Biochemically the essential factor in cataract is the coagulation of the proteins, and many important factors in this process have been discovered in recent years. In general, coagulation of proteins occurs in two stages: (a) denaturation, probably by hydrolysis, whereby the colloidal system becomes more labile; (b) agglutination. Any form of radiant energy—heat, luminous, ultra-violet, radium—can cause coagulation. Ultra-violet rays alter the permeability of the lens capsule (Duke-Elder), diminish the efficiency of the autoxidation system (Adams), and render the proteins more vulnerable to variation in hydrogen ion concentration and salt concentration, e.g., calcium (Burge). Changes in the capsule cause alteration in osmotic pressure and hence in concentration of electrolytes. Deformation of the fibres leads to mechanical strains. Further, the lens proteins are organ specific, especially α- and β-crystallins, but investigations of their serological properties have proved contradictory. The relative parts played by these factors in the development of various types of cataract are obscure, and have not yet led to any satisfactory prophylactic or therapeutic results.

Apart from the experimental production of cataract in animals by various forms of radiant energy it is easily produced in rabbits by administration of naphthalin and other allied poisons: dinitrophenol, for example, used for slimming, has rapidly produced posterior cortical cataract in girls. Dinitrophenol causes a large increase in tissue oxidation. Naphthalene in rabbits is conjugated with cysteine and is excreted as 1-α-naphthylmercapturic acid (Bourne and Young); hence the cataract may be due to depletion of the store of cysteine in the lens. The occurrence of cataract in tetany due to parathyroid deficiency (vide p. 329) when correlated with the excess of calcium in cataractous lenses suggests a definite association with the rôle of calcium in metabolism. Cataract also occurs in the human subject associated with myotonia atrophica and ergotism.

Owing to our ignorance of the pathogenesis of cataract, treatment, apart from steps for minimising the disability, is wholly operative. The type of operation depends largely upon the amount of central sclerosis, *i.e.*, upon the size of the

nucleus (*vide* p. 9). Up to about thirty years of age the nuclear fibres are still fairly soft, and capable of becoming absorbed if the aqueous gains access to them. After this age absorption is very slow and incomplete, and if cataract occurs the nucleus must be removed from the eye. The size of the nucleus then determines the size of the incision which is necessary. The cases in which the nucleus is very small are called *soft cataracts*, since they consist chiefly of soft cortical matter. In most patients over fifty the nucleus is large, and these cataracts are called *hard cataracts*, although the lens is by no means hard throughout. This only occurs in black cataract, in which the nucleus reaches its maximum size, viz., that of the whole lens. Such cataracts require a very large section for their removal. In mature cataracts the brownish appearance of the nucleus by oblique illumination gives some idea of its size and an indication of the size of the section necessary for its removal.

Cataracts are classified according to the position and extent of the opacity or opacities in the lens, and it is found that the situation and distribution correspond with various combinations of clinical conditions—age, general disease, &c. In some cataracts the opacities spread and fuse until the whole lens becomes opaque; such are called progressive cataracts: in others they remain stationary. Senile cataract, which is the commonest form of all, is a progressive cataract.

Senile Cataract. This, as its name implies, rarely occurs in persons under fifty years of age.

In *incipient* senile cataract radial spokes or sectors of opacity are seen, with clear areas between them (Figs. 173, 174). They are difficult to see in daylight or by oblique illumination

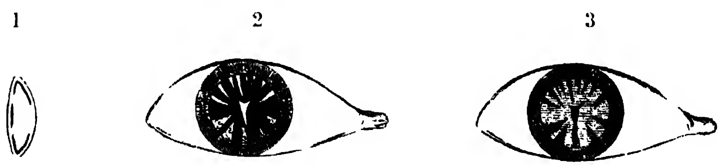


FIG. 173.—Senile cataract. 1, section, showing opacities in the cortex; 2, appearance by reflected light—dark striæ on a red background; 3, appearance by oblique illumination—grey striæ on a dark background. (Nettleship.)

(*vide* p. 95), and cataract should not be diagnosed without confirmation with the ophthalmoscope. With the undilated pupil only the ends of the spokes are seen, but when the pupil is dilated with cocaine or homatropine (*never* with atro-

pine, *vide* p. 285), the linear opacities are often found to be the apices of sectors, with their bases towards the periphery. They generally begin in the lower part of the lens, especially the lower nasal quadrant. Careful examination with oblique illumination and the ophthalmoscope will show that the opacities are in the superficial parts or cortex of the lens, some in front of the nucleus, others behind. They start from the region of the equator and extend towards the axis of the eye, more and more spokes and sectors developing as time goes on.

Seen by oblique illumination the opacities are grey (*vide* p. 95); seen with the ophthalmoscopic mirror at reading distance they appear black against a red background. At the very earliest stage the opacities shift with the incidence of the light, showing that they are merely differences of refractive index (*vide* p. 304). Lens striæ are usually preceded by sectorial alterations in the refractive indices of the lens fibres. These are best seen with the plane mirror, looking alternately light or dark as the incidence of the light is changed. They account for the uniocular polyopia which the patients often notice.

FIG. 174.—Commencing senile cataract, the striæ confined to the lower part of the lens, a very common mode of commencement.

The pupil in old people is seldom so black as in the young, and is sometimes distinctly grey. If the greyness is uniform, cataract should not be diagnosed unless definite opacity is shown on examination with the ophthalmoscope. This greyness without opacity is caused by increase in the refractive index of the cortex of the lens in old people (*vide* p. 53), and is due to increase of reflection and scattering of light.

The above description applies to the commonest arrangement of the opacities in senile cataract; it may be distinguished as the *subcapsular* type. Two other types occur less frequently. In one group the opacities are *supranuclear*. They are more variable in appearance and consist of concentric lines, radial streaks and cloudy patches. This irregularity distinguishes them from lamellar cataract (*q.v.*). In the third group the opacities are *intranuclear*, by which term these cases may be distinguished from the congenital nuclear cataract. In this group the nucleus of the lens is diffusely cloudy, gradually clearing towards the peripheral cortex. Combinations of the various types are not uncommon, sub-

pupil looks black, the brownish colour being revealed only by oblique illumination. The condition is called *black cataract*, though strictly speaking it is not a true cataract. Central intranuclear senile cataracts often show a brownish colour by oblique illumination, due to coincident hyper-sclerosis (*vide* p. 306). They occur more frequently in myopic eyes.

Symptoms. The appearance of black spots in front of the eyes is usually the first symptom complained of. They differ from the ordinary muscæ volitantes occasionally complained of in normal eyes, and much exaggerated in cyclitis, &c., in that they are stationary, retaining their relative position in the field of vision in different positions of the eye. Uniocular polyopia, another symptom, is the doubling, trebling, &c., of the objects seen with the eye. It is due to the irregular refraction of the degenerating lens, so that several images are formed of each object. It is often worse on looking at bright lights, and is therefore noticed most in the evening.

As the opacity extends and becomes denser, the acuity of central vision suffers, especially when there is much central opacity. In the latter cases vision is often better in a dull light, owing to the dilatation of the pupil. In most cases of senile cataract the pupillary region suffers latest, so that a bright light is grateful to the patient, both on account of the better illumination and also because the rays which pass through the irregularly refracting peripheral parts of the lens are cut off by the contracted pupil; but the patients seldom like to face the light.

Eventually the central area becomes affected and vision steadily diminishes until only perception of light remains. In many cases of mature senile cataract fingers can still be counted at a few feet, or at least hand movements discerned. In all cases light should be perceived readily and the direction of incidence accurately indicated. The detection of the *projection of light* is of the utmost importance, as it affords important evidence as to the probabilities of a good result from operation. It is tested as follows. The opposite eye is covered securely by the palm of the patient's hand. Light is then reflected from the ophthalmoscopic mirror into the cataractous eye from various directions, the patient looking straight forwards. He is told to point with his other hand in the direction from which the light seems to come. He ought to do this readily and accurately. If he does not, we suspect some disease of the retina, *e.g.*, patches of retino-choroidal atrophy, &c., and a less favourable prognosis is given. Rela-

tively poor projection is not an absolute contraindication to operation, and each case must be determined on its merits.

It will be seen that cases of advanced cataract in which the fundus cannot be satisfactorily explored by the ophthalmoscope at the first visit, and in which projection is relatively bad, offer considerable difficulties in deciding the treatment to be adopted. It is therefore of the greatest importance that every case of incipient cataract should be most carefully explored and exhaustive notes of the ophthalmoscopic condition taken, so that at a later stage, when the fundus can no longer be observed, its previous condition is already on record. Every case of incipient cataract should therefore have the pupil dilated so that a thorough examination of the eye may be recorded. Homatropine may be used with impunity in most cases, but a drop of 1 per cent. eserine must invariably be instilled before the patient leaves, otherwise there is danger that an attack of glaucoma may be induced by the administration of the mydriatic. If the eye is definitely of glaucomatous type, with small cornea, shallow anterior chamber, &c., cocaine should be used instead of homatropine. The field of vision should also be taken at this stage.

The rate of development of senile cataract varies greatly, sometimes occupying many years, or, indeed, the cataract may never reach maturity. The progress is usually more rapid in very old people. Very rapid maturation in younger patients usually indicates some complication, *e.g.*, cyclitis, diabetes, &c. The forms with fine radial lines are slower than those with cloudy opacities. It is best to examine every case at stated intervals, a careful drawing of the opacities being recorded at each visit. The length of the intervals must be determined by the individual case.

Cataract occurs equally in men and women. It is usually bilateral, but develops earlier in one eye than the other. Cases of hereditary predisposition have been recorded, and in some of these the cataract develops at an earlier age in successive generations (" anticipation," Nettleship).

Pathology. The cubical cells lining the anterior capsule of the lens (*vide* p. 9) undergo vacuolation in senile cataract. Changes in these cells can be made out clinically by using a strong binocular loupe. The cortical opacities are due to the formation of Morgagnian globules and the breaking up of the lens fibres (*vide* p. 305). Cholesterin crystals are not infrequently seen in cataracts.

Biochemistry of Cataract. Although the chemistry of cataract is not fully understood, some of the changes which occur in the cataractous lens are of considerable importance. There is first a diminution in its oxidative metabolism, a decrease in permeability and a loss of the substances active in oxidation—cysteine, glutothione and ascorbic acid. It is not known whether this loss is causal or consequential, but it is true that a cataractous lens is an asphyxiated lens. Chemically there is hydration in the initial stages followed by dehydration later, acidification followed by alkalosis, and gradual replacement of soluble by insoluble proteins. This last is very characteristic, and implies that proteins in an active form are gradually replaced by inactive substances. The coagulation of these proteins is the chief cause of the opacity of cataract. Finally there is an increase of lipoids and an averaging of the concentration of the salts to correspond with that of the blood and the aqueous. The salt content of the normal lens is very different from that of the blood, and as metabolic activity diminishes the two become equalised, but towards the later stages the calcium content becomes very much increased, the salts being deposited in a non-diffusible form, a change which is probably a passive deposition in the inactive tissue.

Senile cataract has been attributed to changes in the cortex produced by shrinkage of the nucleus, but this can be, at most, only a subsidiary factor. Many other theories have been advanced, but in the present knowledge of the biochemistry of the lens they must all be regarded as highly speculative. The definite association of cataract with calcium metabolism in tetany and the raised calcium content of the cataractous lens are strikingly significant facts. In spite of hitherto discordant results the rôle of autocytotoxins, whether of the nature of specific immune bodies or toxins derived from disorder of the general tissue metabolism, demands further consideration.

Treatment. No treatment by drugs, &c., has hitherto proved to have any significant effect upon the progress of uncomplicated senile cataract. Potassium iodide drops, calcium iodide ointment, hormone treatment, especially with parathyroid gland in association with administration of calcium salts, and so on, have been enthusiastically advocated, but are in my experience useless.

Elderly patients frequently have slight peripheral opacities of the same nature as cataract. Much mental anxiety is often caused by telling them that they have "cataract." If cross-examined the surgeon should tell them that they have slight

changes of the same nature as cataract, but that these are quite common in elderly patients and do not necessarily indicate that an operation will be inevitable in the future.

In *incipient cataract* the condition of the patient may be much ameliorated during the tedious process of maturation. A low degree of myopia (1 D to 4 D) may develop during this stage; it is due to relative increase in the index of refraction of the nucleus of the lens, a change in the opposite direction to that which usually occurs (*vide* p. 53). Astigmatism may develop or undergo change. These errors of refraction should be corrected; but often the astigmatism is irregular and glasses afford little help. Considerable loss of vision may be associated with the refractive changes which precede the definite formation of opacities. Tinted glasses may be found beneficial, the tint varying with the circumstances of the case. Amber-tinted glasses are most generally useful. In certain circumstances, *e.g.*, at high altitudes, they cause an extraordinary increase of definition even in normal persons, due to absorption of rays of short wave-length. Blue glasses, which allow the chemically active violet rays to pass, are not contraindicated in this case, since the cutting off of the more luminous rays is restful. These or smoked glasses are indicated, especially when there is a considerable degree of central opacity, since the pupils are kept slightly dilated. For the same reason reading may be much facilitated by isolating only a few lines of the print, the remainder being covered by a black paper mask. The effect may be obtained with greater certainty by instilling a very weak mydriatic. Atropine, $\frac{1}{16}$ to $\frac{1}{8}$ gr. to ℥ i., one drop every morning, may be ordered, if homatropine is found not to raise the tension. The slightest predisposition to glaucoma, *e.g.*, high hypermetropia, small cornea, very shallow anterior chamber, &c., contraindicates this treatment, and it is wise to observe the tension carefully during the treatment in all cases. Sometimes weak atropine causes more blurring, in which case it must be abandoned. Central opacities often cause diminution of central vision apparently out of proportion to the amount of opacity observed.

There is no reason to restrict the use of the eyes in incipient uncomplicated senile cataract, but the patient may be much assisted by instructions as to the arrangement of illumination and so on. If the pupillary area is free, brilliant illumination will be found best; if the opacities are largely central, a dull light placed beside and slightly behind the patient's head will give the best result.

In *mature cataract* the lens must be extracted. Before deciding to operate, attention must be paid to details other than those connected with vision, previously described. The pupil should react promptly and normally to light. Careful search must be made for precipitates on the back of the cornea ("k.p."), for the cataract may be a mature complicated cataract (*vide* p. 325). The urine must be tested to eliminate albuminuria and glycosuria, though these do not necessarily contraindicate operation. The state of the conjunctival sac must be thoroughly examined, and a culture taken. The lacrymal sac is compressed with the finger, so that if there is any regurgitation the secretion from it will be examined. A small cotton-wool swab is rotated in the lower fornix and the secretion thus obtained rubbed over a blood-agar slope. Non-hæmolytic staphylococcus albus and xerosis may be considered innocuous, micrococcus catarrhalis and pneumobacillus doubtful. Staphylococcus aureus, pneumococcus, streptococcus, and such like pyogenic organisms contraindicate operation, which in any case should not be undertaken before forty-eight hours have elapsed lest the very dangerous pneumococcus be overlooked.

If there is the slightest conjunctivitis, and, above all, if there is dacryocystitis, a course of preliminary treatment is necessary. Old people frequently suffer from chronic conjunctivitis induced by senile ectropion, &c. It is best treated by relieving the cause as far as possible, and by the use of astringent lotions. The process of attaining bacteriological cleanliness used to be long and tedious, but the recent introduction of bacteriostatic drugs has largely solved this difficulty. A short course of penicillin drops (1,000 to 2,000 units per c.c.) administered hourly through the day and three hourly during the night for two or three days will usually ensure sufficient surgical cleanliness for the operation to be performed and the wound to heal with safety. In the rare cases where organisms insensitive to penicillin are present the matter is not so simple. An occasional painting with silver nitrate is the most potent means which we possess of removing such organisms from the conjunctival sac in these cases, since they are carried away mechanically with the desquamated epithelium, &c. Irradiation with ultra-violet light and sulphonamides administered by the mouth (*vide* p. 695) have been found efficacious in recalcitrant cases. It is inadvisable to tie up the eye the night previous to operation, for it is found that this procedure favours the growth of bacteria in the conjunctival sac.

The presence of a mucocele is an absolute contraindication to operation. It must be cured (Chap. XXXII.), or the lacrymal sac must be excised, or the canaliculi must be temporarily obliterated. The last may be effected by tying a ligature round each canaliculus or by cauterising each punctum with the actual cautery. The best treatment is usually excision of the sac.

The teeth, nose and throat, and any other likely focus of sepsis, should be examined, and it is very important that any pyorrhœa, &c., should be eliminated before the cataract operation is undertaken.

The treatment of *unilateral* and of *immature cataract* offers some difficulty (cf. p. 323). When the cataract is mature in one eye while the other retains good vision little advantage is gained by operating upon the cataract. The difference in refraction between the two eyes after operation will be so great that it will be impossible for the patient to see well if the refraction is corrected, and if uncorrected the large blurred image formed by the eye may be a positive disadvantage, though it can be relieved by a contact glass. The sole advantage which is gained is an increase of the field of vision on the affected side. This may be a matter of great importance, as in people who work amid machinery or have to go about where there is much traffic; in these exceptional cases extraction is indicated. There is also the slight advantage that the eye is prepared for the time when vision fails in the less affected eye, but this may be long delayed. These slight advantages do not as a rule justify operation, which, it must be remembered, is attended with some, if usually trivial, danger, not only to the eye operated upon, but also to the other eye (*vide* p. 464). On the other hand, the cataract must not be allowed to progress to too advanced a condition of hypermaturity. Operation is then more difficult and more dangerous. The case should be watched, and if signs of thickening of the capsule, calcareous deposits, &c., appear, extraction should be performed even though the vision in the other eye is still good.

Cases of immature cataract with loss of useful vision require even more skill in the determination of the best time for operation. The difficulties and dangers of extraction are undoubtedly increased by operating while there is still a large amount of clear soft cortex. It is difficult to remove from the eye, tends to the production of iritis and other complications, and leads to the formation of dense secondary cataract (*vide* p. 324). Immaturity, however, is not an absolute bar to

operation; extraction under these circumstances may be followed by excellent results, especially if performed by the intracapsular method. It must be remembered that the patients are old, and, if not operated upon, are doomed to practical blindness, which in the lower classes entails the loss of all wage-earning capacity. Operation will probably be attended by at least the recovery of useful vision, whereby the conditions of existence are much ameliorated. It is not necessary, therefore, to wait indefinitely for complete maturity if useful vision has already been lost.

Some surgeons temporise in these cases, performing a preliminary iridectomy, on the grounds that the operation accelerates the ripening of the cataract. It certainly occurs in rare cases, but is by no means constant. Preliminary iridectomy has the advantage of facilitating the subsequent extraction, but has the considerable disadvantage of subjecting the patient to the discomforts and dangers of two operations instead of one. It may be employed in complicated cases in which it is desired to test the reaction of the eye to operative interference, for it is a less severe operation than extraction, and will afford indications as to the advisability of further procedures. It is also to be advocated in cases with much diffuse opacity, often more concentrated in the central part of the posterior cortex, for in them the diminution in visual acuity is very marked, maturation is indefinitely delayed and the complications produced by a large amount of sticky cortex are much to be feared. The extraction of the cataract should not be performed until at least four weeks after the preliminary iridectomy.

Preliminary iridectomy is indicated most definitely in cases of cataract with increased intraocular tension. The tension may be raised owing to the swelling of the lens in the incipient stage, in which case iridectomy usually relieves the pressure. Nearly mature cataract may be associated with increased tension. It might be thought that an ordinary combined extraction would relieve both conditions, but these cases do not usually progress smoothly. It is much better to do a preliminary iridectomy, extraction following after the usual interval.

In some cases eyes with incipient cataract have been trephined for chronic glaucoma (*vide* p. 301) or cataract has developed subsequent to trephining. Theoretically it is obviously objectionable to make a cataract incision through the trephine hole, though such cases often do well—possibly because extraction may render an eye less prone to the usual form of chronic glaucoma. It has been sug-

gested to extract downwards, but it is probably wiser to make the upper part of the incision in the cornea in front of the trephine hole.

The correction of the refraction after extraction of cataract is dealt with elsewhere (*see* Aphakia, p. 533).

Cataracts of Congenital or Infantile Origin. These are almost always partial and stationary. The commonest forms

FIG. 177.—Lamellar cataract. 1, 2, 3, as in Fig. 173. (Nettleship.)

are lamellar and anterior capsular; less common are the various forms of congenital cataract, mostly of lamellar type, sometimes central or total.

Lamellar Cataract (*Syns.—Zonular, Perinuclear Cataract*). This usually occurs so early in infancy that it is doubtful if it is not congenital. Although there is no true nucleus at

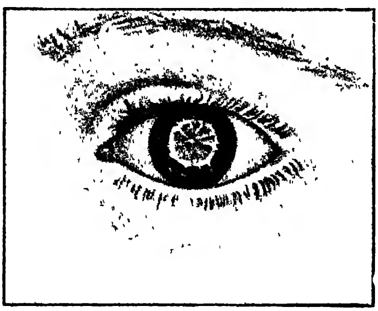


FIG. 178.—Lamellar cataract.

FIG. 179.—Lamellar cataract with very slight

this early age the central parts of the lens are conveniently termed the nucleus. The opacity in lamellar cataract is situated in the layers surrounding this central core, which itself usually contains punctate opacities; the superficial cortex is quite clear (Figs. 177—179). When the pupil is dilated a grey discoid opacity is seen, surrounded by a perfectly transparent marginal area. The diameter of the disc varies, that of the clear peripheral area varying inversely. With the

mirror the disc appears black and sharply defined at the outer edge, diminishing in density towards the centre; the peripheral area shows a normal red reflex. Along the outer edge spokes of opacity, resembling the handles of a steering wheel, often extend slightly into the clear area (Fig. 178). They are called riders, and are due to spindle-shaped opacities between the lens fibres in layers a short distance outside the main opacity. Occasionally two concentric rings of opacity are seen. The cataract is usually stationary until late in life, but cases occur in which total opacity gradually develops. Both eyes are almost always affected, though not always to the same degree.

The opacity is always sufficiently large to fill the area of the undilated pupil. The diminution of vision is therefore entirely dependent upon the density. The patient is brought for examination on account of defective vision. He usually holds objects very close to the eyes and is thought to be "short-sighted." Myopia is indeed not uncommon in these cases, but the approximation of objects is usually for the purpose of obtaining larger retinal images (*vide* p. 37).

FIG. 180.—Hypoplasia of teeth.

There is no doubt that lamellar cataract is due to a period of mal-nutrition at some stage of late intra-uterine or early infantile life. It has been found that lamellar cataract in young rats, offspring of mothers fed immediately after birth of the litter on diet lacking in vitamin A, fat, and phosphorus, may become normal if the food is replaced by a normal diet. No rachitic changes are found in the bones, whereas older animals on a diet poor in vitamin A get rickets under these conditions, but no lens changes. Epithelial structures are most affected. Epithelium as a rule covers surfaces, and the oldest cells are cast off, being replaced by young cells derived from the basal layer. There are two sites in which the epithelium persists: (1) the lens, where, owing to its formation as an invagination of epiblast, the oldest cells are central and cannot be cast off; (2) the enamel of the teeth, where the cells become calcified and thus retained. Hence lamellar cataract is almost invariably accompanied by defective enamel in certain of the permanent teeth. The hypoplasia differs essentially from the condition of the teeth in congenital syphilis. The teeth have an eroded appearance, with transverse lines across them, the incisors and canines being most affected (Fig. 180).

The time at which the pathological process took place is indicated by the size of the diameter of the opacity and the particular teeth affected with hypoplasia. Only those teeth are affected whose enamel germs are being formed at the time. As regards the lens, the youngest fibres are the most superficial (*vide* p. 9), so that the diameter of the opacity indicates the size of the lens at the time. The usual diameter of the opacity and the particular teeth affected both indicate that the malnutrition generally occurs at about the time of birth or shortly afterwards. Concentric rings of opacity are accounted for by successive periods of malnutrition.

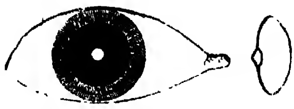


FIG. 181.—Anterior capsular cataract. (Nettleship.)

The cause of the malnutrition is probably to be found in errors of feeding and possibly exanthemata. It is probable that one cause is the occurrence in the mother of rubella during the earlier part of pregnancy and it is possible that other virus infections act similarly. There is some reason to think that rickets is a cause, and congenital syphilis has been indicted, but on insufficient grounds. A history of convulsions is very common.

Treatment depends upon the density and the diameter of the opacity. In cases with dense opacity and very poor vision with undilated pupils the treatment depends upon the diameter of the opacity. If it is small with a wide area of clear cortex, and if distant vision is much improved when the pupil is dilated and the refraction corrected so far as possible, an optical iridectomy may be performed. In some cases, however, the opacity increases, so that iridectomy should not be done until there is fair certainty that the condition is stationary. Suitable cases for this operation are therefore quite uncommon. Usually the opacity is large, and it is then necessary to remove the lens, which has the grave disadvantage that it abolishes accommodation. Since the patients are almost always seen when quite young, the central core of the lens does not yet form a hard nucleus. Non-sclerosed lens fibres become absorbed if the aqueous gains access to them. Hence lamellar cataract can be treated by discission or needling, whereby an aperture is made in the anterior capsule through which the aqueous enters. This is the ordinary treatment of lamellar cataract, but it should not be employed unless the vision is seriously impaired

or the other methods of treatment are impossible. As all varieties of density are met with the advisability of needling in cases with fair vision has to be considered. The decision of this question depends upon whether vision with corrected refraction and retained accommodation is to be preferred to probably improved vision after operation without accommodation. I am of the opinion that vision of 6/12, or even 6/18, with retained accommodation, is more valuable than a problematic 6/9, or even 6/6 without accommodation, but with the necessity of wearing constantly very strong convex glasses for distance and still stronger ones for near work. I do not therefore operate in such cases.

It is not advisable to needle lamellar cataracts until the child is 9 or 10 months old. During the waiting period the pupils should be kept dilated with 0·5 per cent. atropine once

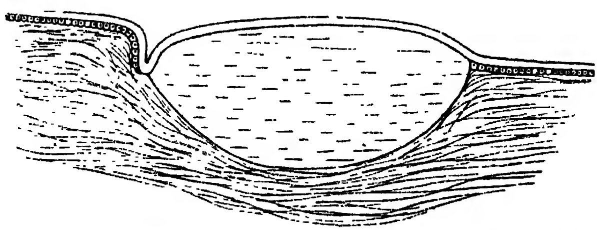


FIG. 182.—Diagram of section of anterior capsular cataract.

every other day, so that the retina may be stimulated by light passing through the clear zone. This procedure diminishes the risk of nystagmus or squint developing.

Anterior Capsular Cataract (*Syn.—Anterior Polar Cataract*). This form of cataract is commonly known as anterior polar. It is best to reserve this term for any cataract at or near the anterior pole of the lens, since there are two forms of anterior polar cataract, viz., anterior capsular and anterior cortical. Similarly the term posterior polar should be used in the same manner, though there is more ambiguity here. There are two forms of opacity which are known as posterior polar cataract. The posterior cortical cataract is the commonest form of complicated cataract (*vide* p. 325) (Fig. 184). The other posterior polar "cataract" is not strictly a cataract at all, since it is due to persistence of part of the posterior vascular sheath of the lens, and is therefore situated upon the posterior surface of the lens, *i.e.*, it is not a true lenticular opacity (Fig. 186), though there is always some opacity in the adjacent lens fibres.

Anterior capsular cataract is due to abnormal proliferation of the cubical cells which line the anterior capsule (Fig. 182), and is usually limited in uncomplicated cases to a small area in the centre of the pupil (Fig. 181). The stimulus to proliferation is caused by contact with the normal or inflamed cornea. Contact of the lens with the normal cornea causes opacity in the lens only if it occurs at an early age. This is indeed fortunate, for if it were not so many intraocular operations, *e.g.*, iridectomy, would be impossible. In very young children it is probable that a very short time of contact is all that is necessary. The older the patient the longer is the time required. Contact with an inflamed cornea is more liable to produce an anterior capsular cataract than with the normal cornea. In most cases it is due to perforation of a corneal ulcer, usually caused by ophthalmia neonatorum ; more rarely to a perforating wound.

Anterior capsular cataract is sometimes congenital, when it is also probably due to contact with the cornea, possibly owing to delayed formation of the anterior chamber without actual perforation. In these cases it is almost always bilateral, whereas the acquired form is generally unilateral.

When an ulcer perforates, the aqueous escapes and the lens and iris are driven forwards into contact with the back of the cornea. If the perforation becomes blocked with iris the anterior chamber re-forms, the length of time of contact between the lens and cornea varying in different cases. If it is short, no harm may be done to the lens unless the patient is very young. If it is more prolonged, an anterior capsular cataract is formed and the lens adheres more or less to the wound. When the anterior chamber re-forms, the lens usually separates completely from the cornea : less frequently the adhesion stretches out into a fine filament, which may persist or break. Occasionally the adhesion is so firm that the lens is permanently anchored to the cornea : the eye is usually lost by panophthalmitis or secondary glaucoma in these cases.

The dragging upon the adhesion when the anterior chamber is re-formed may cause a conical protrusion of the cataract—*pyramidal cataract* (Figs. 116, 181).

The deleterious effects of contact may affect the underlying cortical fibres, so that an anterior cortical cataract may occur with a capsular one. The cubical anterior capsular cells may grow in between the capsular and cortical opacities. They give rise later to normal transparent lens fibres, so that the two opacities become separated by a narrow clear zone of cortex. In the absence of cortical degeneration the opacity is

usually so small and sharply defined that vision is little impaired, and no treatment is required.

Congenital Cataract manifests itself in a variety of different forms. As already stated, *anterior capsular* and typical *lamellar cataracts* may be congenital. Many other forms are nearly allied to the lamellar type, those occurring early in fœtal life being small in diameter (*vide* p. 319). To this category belong the following :—

Central or nuclear cataract, a small spherical opacity in the centre of the lens, surrounded by clear cortex.

Fusiform cataract, also called spindle-shaped, axial, or coralliform, an antero-posterior spindle-shaped opacity, sometimes with offshoots giving an appearance much resembling coral : this form shows a great tendency to occur in families. *Discoid cataract* is also a familial form, showing a somewhat ill-defined disc of opacity just behind the nucleus in the posterior cortex.

In other congenital cataracts minute points of opacity are seen scattered throughout the lens, or limited to parts—*punctate cataract*. Many varieties of this type occur. Bluish spots, seen by oblique illumination near the anterior surface of the lens, and of congenital origin, are not uncommon. They are almost transparent when viewed with the ophthalmoscope, remain unchanged throughout life and require no treatment. A single minute round opaque spot, usually eccentric, and situated on the back of the lens, is not infrequently seen in the routine examination of patients with the ophthalmoscope. This spot can always be found with the slit-lamp, and is caused by the remains of the fœtal posterior vascular sheath of the lens.

Most of these congenital cataracts are stationary. They may be associated with other congenital stigmata, such as nystagmus (*vide* p. 566), congenital colobomata, &c. They may require no treatment, or optical iridectomy or discission may be indicated (*vide* p. 319). It is wise to wait until puberty in cases apparently suitable for optical iridectomy, the pupils being kept dilated with atropine in the meantime, since some are not stationary but gradually progress to the formation of total cataract.

Total cataract may be congenital or the result of progressive partial congenital cataract. The lens may be shrunken and much degenerated, and there are often other congenital defects in the fundus, &c. These cases should be treated by discission, but the prognosis given must be very guarded. Sometimes

needling reveals persistence of the posterior vascular sheath of the lens, with or without persistence of the hyaloid artery. In such cases violent attempts to remove the opacity by needling will result in the loss of the eye.

The pupil is often small with congenital total cataract and reacts very feebly, if at all, to light and on convergence. Neither does it dilate appreciably with atropine. Attempted discission of the shrunken cataract often causes rupture of suspensory ligament, and it may be necessary to remove the membranous lens with toothed capsule forceps (Fig. 129) through a keratome incision. Vitreous is usually lost and there may be severe reaction, but the result not infrequently justifies the heroic measures.

Congenital cataracts should not in general be needled until the child is 9 or 10 months old. If there is a clear peripheral zone they should be treated like lamellar cataracts (*vide* p. 319). In cases where the lens is completely opaque, or the pupil will not dilate, and when squint or nystagmus is developing, it is advisable to needle at a much earlier age, though there is some risk in doing so.

The Treatment of Unilateral Cataract in Children. When dense unilateral cataract occurs in a child, whether from lamellar or congenital cataract or from traumatism (*vide* "Traumatic Cataract"), so that the pupil becomes grey or white, it is advisable to needle early. In these cases the appearance of the eye militates against the individual obtaining employment. Moreover, needling is a less severe operation than extraction of cataract, which may become necessary at a later date if the other eye fails. The treatment of unilateral cataract in children differs therefore from that of a similar condition in adults (*vide* p. 315).

Coronary Cataract represents the same type of developmental cataract as is seen congenitally, but since it develops at or soon after puberty it is situated in the deep layers of the cortex and the most superficial layers of the nucleus. The corona of club-shaped opacities is usually hidden by the iris while the axial region and the extreme periphery of the lens remain free. The opacities are not progressive and do not lead to complete opacification of the lens: their importance lies in their recognition as a developmental anomaly, for if they are seen when the pupil is dilated and their character is not recognised, the examiner may be led to diagnose a progressive cataract in a young adult.

Secondary Cataract (*Syn.—After-cataract*) is the opacity which persists or follows after the extraction or discission of the lens.

In both these operations the posterior and part of the anterior capsule remain *in situ*. If only the posterior capsule remains in the pupillary area the corrected vision will probably be good, though it may be much impaired by wrinkling of the capsule and the consequent irregular refraction. In many cases, especially when the cataract is not quite mature, some soft, clear cortex sticks to the capsule. This becomes partially

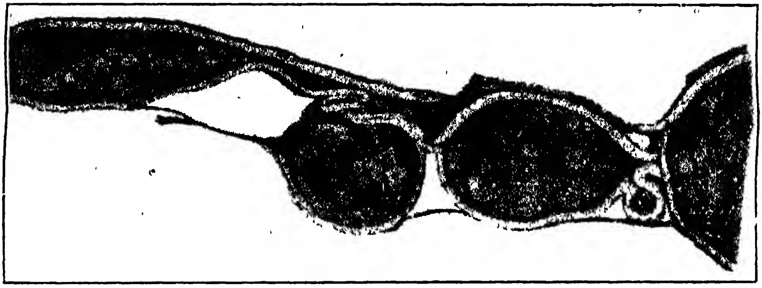


Fig. 183.—Secondary cataract from a section by Treacher Collins.

absorbed by the action of the aqueous, but it often becomes shut off from the aqueous by adhesion of the remains of the anterior capsule to the posterior capsule. In such cases the cubical cells which line the anterior capsule also persist; they continue to fulfil their function of forming new lens fibres, though those formed under the abnormal conditions are abortive and opaque (Fig. 183). If these remnants lie in the

Fig. 184.—Posterior cortical cataract. 1, 2, 3, as in Fig. 173. (Nettleship.)

pupillary area a dense membrane is formed through which the rays of light penetrate with difficulty, so that vision is very imperfect. If the previous operation has been followed by iritis, exudates also adhere to the lens remnants and organise, thus contributing a fibrous membrane in addition.

Secondary cataract is demonstrated either by oblique illumination or by the ophthalmoscope. If fine, it may be difficult to see, forming a grey film by the former method, a

cobweb-like haze by the latter. The denser membranes are easily recognised. They vary in density, showing coarse opaque bands separated by more transparent areas.

Treatment. Secondary cataract requires needling (*vide* p. 485).

Complicated Cataracts (*Syn.—Secondary cataracts*) are those forms which result from malnutrition of the lens, due to disease of other parts of the eye or of the general system. The lens is nourished by lymph which is supplied by the ciliary body. If, owing to disease of the ciliary body or to lymph secreted from abnormal blood, the nutrition of the lens suffers, opacities are formed. They usually commence in the centre of the posterior part of the cortex, and are therefore at first *posterior cortical cataracts* (often called posterior polar) (*vide* p. 320) (Figs. 184, 185). The opacity seldom remains confined to this situation: it progresses, affecting first the periphery of the anterior cortex close to the nucleus, finally involving the whole cortex. In many cases the opacities are fine and dust-like, and are scattered throughout the cortex from the commencement, increasing in number and density as time goes on.

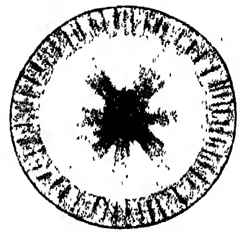


FIG. 185.—Posterior cortical cataract (posterior polar cataract), seen by reflected light.

The total cataract formed in this manner is usually soft and uniform in appearance. In still later stages the watery constituents become absorbed, the capsule becomes thickened, the whole lens shrinks, giving rise to tremulousness of the iris, and other degenerative changes—calcification, &c.—ensue.

Complicated cataracts occur in advanced cases of cyclitis, in absolute glaucoma, in choroido-retinitis—disseminated choroiditis, retinitis pigmentosa, &c.—in high myopia, in detachment of the retina, &c.; they also occur in suppurative inflammation of the cornea, especially that produced by ulcus serpens. The opacity in the posterior cortex, which is generally stellate in shape, is seen in its most characteristic form in retinitis pigmentosa, in which disease also its slow progress can be easily watched. The vision is already much diminished before complicated cataract makes its appearance. This fact is of the utmost importance from the prognostic point of view, since, even if the cataract is successfully removed, the progressive diminution in vision due to changes in the fundus is

not thereby influenced. In every doubtful case not only must the central and peripheral vision be carefully investigated, but an exhaustive search must be made for precipitates upon the back of the cornea.

Treatment must be directed in the first case to the cause of the complication. This is often tedious and unsatisfactory, but must be persevered in as long as useful vision persists. If then the perception and projection of light appear to be fairly good, and the cataract is of a nature suitable for operation, it should be removed by discission or extraction, according to the age of the patient. Many cases are not suitable for

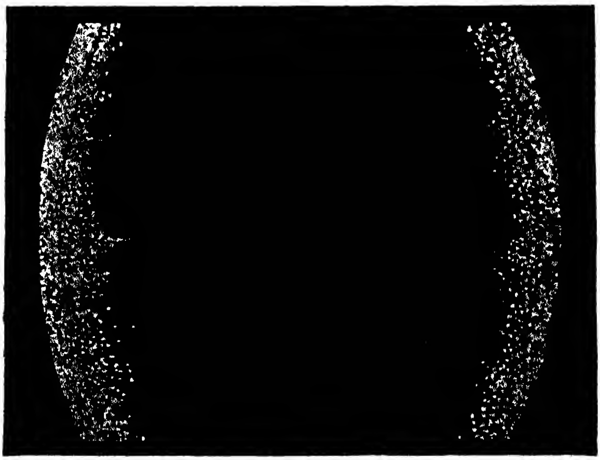


FIG. 186.—Diabetic cataract. Optical section as seen with the slit-lamp. (Goulden.)

operation, mostly on account of cyclitis or the very defective vision and projection of light. Even in these, if there is a possibility of success, operation may be undertaken after warning the patient of the doubtful issue, for the loss of such an eye weighs little against a reasonable probability of improved vision. It is wise in these cases to do a preliminary iridectomy (*vide* p. 316).

Diabetic Cataract should be regarded as a form of complicated cataract. Cataract in diabetic persons is by no means always diabetic in the proper sense of the term. Senile cataract of the usual type, following the usual course, often occurs, and should be treated in the ordinary manner, though in the early stages the general disease must invariably receive

every attention, both in the matter of diet and drugs. True diabetic cataract is comparatively rare, and occurs in younger adult patients. It is always bilateral, and commences with small discrete cloudy opacities immediately beneath the anterior and posterior capsules (Fig. 186). Dusty opacities then appear throughout the cortex and rapidly increase until total cataract supervenes. A uniform milky opacity of rapid onset should always suggest the possibility of diabetes, though of course the urine should be tested as a matter of routine in all cases of cataract.

Diabetic cataract, though usually occurring in patients with a large percentage of sugar in the urine, is not immediately due to the mere presence of sugar in the aqueous, for it is never sufficiently concentrated to cause cataract *per se*. Nevertheless, there can be little doubt that osmotic changes are a prominent factor in the pathogenesis and account for the accumulation of droplets beneath the capsule.

Cataract is readily produced in rats by a diet containing large doses of lactose or galactose (but not dextrose). These sugars are said to aid absorption of calcium from the intestine, and it is noteworthy that there was an increase of calcium in the eyes with cataract, but no increase in the blood.

Treatment. It is imperative in all cases of true diabetic cataract to treat the general condition before adopting operative measures. It is rare for the opacity to clear up under such treatment; but since cases do occur, (Nettleship), and since operations upon the eyes of diabetic patients have special dangers of their own, this chance should always be afforded.

If general treatment is unsuccessful the cataract must be extracted. In spite of the special difficulties attending the operation the results are often quite satisfactory, though a guarded prognosis should be given. Contrary to what might be anticipated, the wound usually heals well. The special dangers are local and general. Of the former, the tendency to severe iritis and to hæmorrhage are the most important. The necessary manipulation of the iris is likely to set up traumatic iritis of a peculiarly violent type. Iridectomy in the course of the operation may be attended with much hæmorrhage, which not only obscures the view of the field of operation, but may endanger the eye: violent intra-vitreous or subchoroidal hæmorrhage may destroy the eye at the time of operation.

Hence it is desirable to remove a diabetic cataract by simple extraction, *i.e.*, extraction without iridectomy, or at any rate with only a peripheral buttonhole iridectomy (*vide* p. 502). The chief danger affecting the general system in these cases is the risk of the sudden onset of diabetic coma. It is comparatively slight, and must be guarded against as far as possible by a suitable course of anti-diabetic treatment before operating. Another grave danger is that of septic infection. If it occurs, panophthalmitis rapidly supervenes, owing to the very defective resistance of the tissues.

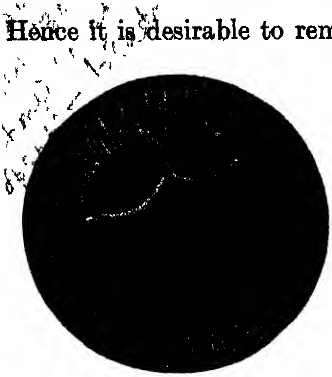


FIG. 187.—Coloboma of the lens (Marcus Gunn). The direction of the coloboma, upwards, is somewhat unusual. Note the defective development of the suspensory ligament of the lens.

Traumatic Cataract. See p. 450.

Persistence of the Posterior Vascular Sheath (*Syn.—Posterior Polar Cataract*). See p. 334.

Glass-workers' Cataract occurs chiefly in men who have long been engaged in glass manufacture, particularly beer bottles and plate glass. It does not affect those who make flint-glass bottles or pressed glass articles, the heat of the furnaces being much less in these cases. The cataract is very characteristic. In the early stages there is a small disc of opacity in the posterior cortex of the lens, thinner and more sharply defined than the posterior cortical opacity of complicated cataract, but it may extend throughout the cortex in the later stages. The radiations from the molten glass contain few ultra-violet rays, which are therefore probably not the cause. It is most likely that heat is the cause, acting, not directly on the lens, but upon the iris and ciliary body, thus influencing the nutrition of the lens. As already stated (p. 23), heat radiation is absorbed by the pigment epithelium of the iris, ciliary body, and retina. Vogt has produced cortical cataract in rabbits by exposure to radiation of wave-length 670—700 $\mu\mu$ for less than an hour.

It has recently been shown that certain *iron-workers,* especially tin-plate millmen and chain-makers, suffer from an identical condition. It is apparently rare in other iron-workers, probably because they seldom look at the hot metal, and when doing so protect their eyes with coloured glass. It does not occur among acetylene welders, &c., who are unable to carry out their work

without efficient protection of the eyes by suitable deeply tinted protective goggles.

Irradiation Cataract, due to exposure of X-rays or radium, resembles the early posterior cortical stage of glassworkers' cataract. One course of deep X-rays may suffice. There is a considerable latent period, which may be at least two years. Only the γ-rays of radium seem to be nocuous.

Perinuclear cortical opacities occur in post-operative *tetany*: in parathyroidectomised animal tetany and lens opacities coincide with periods of Ca deficiency in the food. Similar opacities also occur in cases of *Mongolian idiots* at about puberty and *myotonia atrophica* (*vide* p. 601).

Dislocation of the Lens. See p. 441.

Congenital Abnormalities of the Lens

Besides the various forms of *congenital cataract* (*vide* p. 322), abnormalities in the shape and position of the lens occur, often associated with other malformations of the eye.

Coloboma of the lens is the condition in which there is a defect in the inferior margin, usually notch-shaped; less frequently it occurs in some other part of the margin (Fig. 187). It is due to defective development of part of the suspensory ligament.

Ectopia lentis or congenital dislocation is a subluxation of the lens, usually upwards or up and in, and bilateral. The condition is often hereditary. The lens is small, but the edge is generally invisible until the pupil is dilated. The usual signs of subluxation (*vide* p. 441) are then seen. It is sometimes associated with arachnodactyly.

Lenticonus, generally posterior, is an abnormal curvature of the lens, so that the surface is somewhat conical instead of spherical. It is best studied by means of the slit-lamp.

CHAPTER XVI

Diseases of the Vitreous

The vitreous humour is an inert, jelly-like structure which subserves optical functions (*vide* p. 10). It contains proteins and a muco-protein. It has all the properties of a hydrophilic gel, undergoing turgescence in an alkaline, deturgescence in an acid aqueous medium. It is in very unstable equilibrium, and readily becomes transformed into a sol, either by mechanical means or chemical, *e.g.*, metabolic changes. Hence, "fluid" vitreous is a common pathological condition. It possesses no blood vessels in post-natal life, and is incapable of becoming inflamed : the old term "hyalitis" rests upon misconception, and should be avoided. We have, therefore, to deal only with symptomatic conditions.

Opacities. Black specks, floating before the eyes, are seen by normal persons under favourable conditions. These *muscæ volitantes* are opacities of various kinds, viewed entoptically, *i.e.*, they throw a shadow upon the sentient elements of the retina, thus appearing as dark spots in the field of vision. Any relatively intransparent bodies situated anterior to the rods and cones are therefore able to produce muscæ. To this category belong the corpuscles circulating in the retinal blood vessels ; if it were not for the fact that the retina is normally adapted for red light the entoptic images of the circulating corpuscles would be a serious impediment to clear vision. Other muscæ are due to minute specks in the vitreous, so small and so slightly intransparent that they cannot be seen objectively by the ophthalmoscope.

Under abnormal conditions muscæ may be so increased as to interfere with vision and to become visible by the ophthalmoscope. They then indicate some disease of the uveal tract, particularly of the ciliary body : they are found in cyclitis, retino-choroiditis, myopia, &c. In their slightest manifestation they are dust-like opacities, which may permeate the whole vitreous or be limited to the anterior part. When very fine a plane mirror and magnification by a convex lens are necessary in order to distinguish them (*vide* p. 110). They are due to minute albuminous coagula and aggregations of leucocytes, the former derived from the ciliary body and choroid,

the latter only from the ciliary body and possibly the retina. In the more severe cases flakes and threads are seen, and the entoptic images may be so sharply defined that an intelligent patient is able to draw them accurately. The larger opacities are often found after hæmorrhage into the vitreous. They almost invariably float about, showing that the vitreous is fluid (*vide infra*), though they may be more or less anchored to the retina. Vision is often best in the morning, before the muddy vitreous has been stirred up by movements of the eyes.

Dense vitreous opacities obscure the view of the fundus with the ophthalmoscope. In moderate cases the disc and vessels may be made out, as if seen through a dense haze. The disc looks redder than usual, and it may be difficult to decide whether there is papillitis or not.

Very frequently in the slighter cases no objective signs of disease can be made out in the fundus; the foci are either too fine to be appreciated or are anterior to the field of ophthalmoscopic vision, *i.e.*, in the anterior part of the choroid or in the ciliary body.

Treatment. Slight cases of muscæ volitantes, without objective signs, require no direct treatment. Patients should be advised to ignore the spots as much as possible, as they are often only visible when attention is specially directed to them. Any error of refraction should be corrected, and special indications as to the amount and conditions of near work should be given. In many cases the patients suffer from gastro-intestinal disorders, which should be suitably treated.

Treatment of the more severe cases of vitreous opacity depends upon the cause. When this is known, as in iridocyclitis, syphilitic retinitis, retino-choroiditis, tubercle of the uveal tract and so on, attention must be specially directed to the treatment of the primary foci. The prognosis is best in the syphilitic and the milder iridocyclitic cases.

When the cause cannot be discovered iodides are generally given, as they are supposed to promote absorption. They may be combined with mercury, even in non-syphilitic cases.

The eyes should be kept at rest with atropine and dark glasses. Hot bathings and leeches or dry cupping may be tried; and subconjunctival injections have been beneficial in some cases, but must not be used if there is any active inflammation. Dionin has also been advocated.

Fluidity of the Vitreous (*Syn.—Synchisis*, συγχέω, to pour together, disturb) is due to absorption of the fibrils and

and possibly tuberculous subjects, minute bunches of new-formed blood vessels project from the disc or retina, usually near the disc, into the vitreous (*vide* p. 363)

Pus in the Vitreous. This is found only in panophthalmitis, which is almost invariably due to a perforating wound or ulcer, though cases of metastatic inflammation of the retina and choroid also occur and lead to a similar result (*vide* p. 343). The reflex with the ophthalmoscopic mirror is poor or absent. Oblique illumination shows a yellow mass behind the lens. The eye is always intensely inflamed, and little difficulty is usually experienced in arriving at a correct diagnosis. The *treatment*

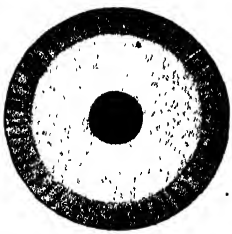


FIG. 188.—Opacity due to persistence of part of the posterior vascular sheath of the lens, often called posterior polar cataract. Note that the opacity is usually more circumscribed than in posterior cortical cataract (Fig. 185).

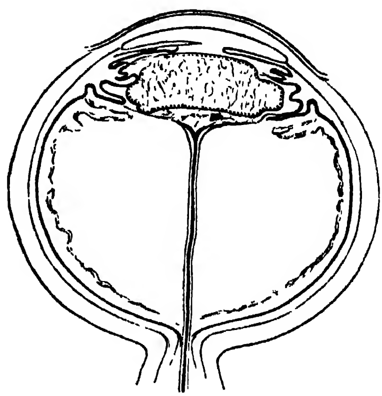


FIG. 189.—Diagram of persistent hyaloid artery, with persistence of the posterior vascular sheath of the lens. (Treacher Collins.)

in cases of exogenous infection is that of panophthalmitis due to perforating injury (*vide* p. 462). In the rarer cases of endogenous infection it is that of the primary cause.

Persistent Hyaloid Artery. The hyaloid artery, which in fœtal life nourishes the vitreous and supplies blood to the posterior vascular sheath of the lens, becomes obliterated during the sixth and seventh months, and usually disappears completely before birth. A remnant, stretching forwards from its origin on the disc, is normal in oxen. Similar remnants are not very rare in man: they appear as a strand projecting from the disc into the vitreous. There may be membranes veiling the disc from view in such cases. A remnant of the anterior part of the hyaloid artery can be seen in normal

eyes, with the slit-lamp, as a whitish coiled strand attached to the posterior capsule 1 to 2 mm. to the nasal side of the posterior pole. Sometimes a larger portion persists, appearing as a circular spot on the back of the lens, often inaccurately termed a *posterior polar cataract* (Fig. 188). More or less of the posterior vascular sheath may persist in such cases, giving rise to a larger central opacity on the back of the lens. In these cases there is always a gap in the posterior capsule of the lens and the adjacent posterior cortical lens fibres are cataractous. The hyaloid artery may persist in its entirety with more or less of the vascular sheath (Fig. 189). It may contain blood, and blood vessels may be seen upon the back of the lens; it is then liable to be mistaken for a glioma of the retina, and constitutes one form of pseudoglioma (*vide* p. 430).

The appearance of the posterior part of the hyaloid artery, when persistent, is very characteristic. A filmy, greyish cord, sometimes containing blood, passes forwards from the disc towards the lens. It undergoes serpentine writhings when the eye is moved.

When the hyaloid artery is persistent there is often defective or atypical development of the vitreous, part of which resembles fibrous tissue in structure. Every stage may be met with, from that interfering little or not at all with vision to complete opacity, usually with maldevelopment of the whole eye—microphthalmia.

Foreign Bodies in the Vitreous. See p. 456.

Parasites in the Vitreous. *Cysticercus* is very rarely found in the vitreous in England, though it is less uncommon in some other countries. The actual parasite may be seen ophthalmoscopically as a pearly translucent mass with peristaltic movements. The treatment—removal—is very difficult.

CHAPTER XVII

Diseases of the Choroid and Retina

It has already been pointed out that different parts of the uveal tract rarely suffer alone. This intimate connection is most marked in the anterior parts, but clinically the evidence of cyclitis when the choroid is inflamed is slight, though not entirely wanting. The outer layers of the retina are dependent for their nutrition upon the choroid, so that when the latter suffers the former is always involved secondarily. Primary affections of the retina may occur without involvement of the choroid : primary affections of the choroid invariably involve the retina secondarily in greater or less degree.

This profound relationship between the retina and choroid, so indisputably manifested clinically, renders it advisable to consider their diseases in close connection with each other. It will be found that some diseases commonly designated as " retinitis " are in reality secondary to a primary choroiditis, while others also included under the same term are not inflammatory, as is suggested by the word. It is well, therefore, to bear in mind that " retinitis " is used in a broad sense, and might in many instances be replaced with advantage by the term "retinopathy." The same ambiguity is noticed in the use of the term " choroiditis," which frequently designates a degenerative condition without any evidence of inflammation.

Primary Affections of the Choroid

Vascular Disorders. Although the blood-supply of the uveal tract is almost entirely derived from the posterior ciliary arteries the peculiar distribution resulting in the formation of the circulus arteriosus iridis major causes involvement of both iris and ciliary body in pathological vascular conditions, whereas choroidal lesions are often restricted to isolated areas. Sclerotic changes in the choriocapillaris, for example, may be sharply delimited, as in macular degeneration, &c., and doubtless many of the patches of so-called choroiditis and choroidal degeneration are of vascular origin. Some are probably due to embolism or thrombosis, but have rarely been proved so by histological examination. Localised choroidal hæmorrhages occur, but are difficult to diagnose from the rounded retinal hæmorrhages posterior to the vessels (*vide* p. 360). Massive

hæmorrhages from the choroid occur in expulsive hæmorrhage (*vide* p. 503)

Inflammation affecting the choroid primarily—*choroiditis*—occurs in two forms, exudative and suppurative. The former appears in the form of isolated foci of inflammation scattered over certain areas of the fundus, and is conveniently classified according to the position of the areas involved. The latter spreads over the whole choroid and retina, and the primary seat may be in the retina: it leads ultimately to panophthalmitis.

Exudative choroiditis is often syphilitic in origin, though certainly not so generally as was formerly thought; it affects chiefly either the posterior part of the fundus—disseminated choroiditis—or the anterior part—anterior choroiditis.

Disseminated Choroiditis may be taken as a type of the disease (Plate IX., Fig. 1). The recent foci are seen ophthalmoscopically as round yellowish spots; when near a retinal vessel they lie at a deeper level than the vessel. They are due to infiltration of the choroid, the exudates hiding the choroidal vessels which cause the normal red reflex. In the early stages the elastic membrane of Bruch is intact; in these circumstances only fluid exudates can pass through it, but these suffice to make the overlying retina cloudy and grey. Hence the edges of the spots are a little hazy and ill-defined. The exudates not only pass into, but also through the retina, so that punctate or diffuse opacities are seen in the vitreous. When the vitreous haze is excessive the ciliary body is also probably involved. In later stages the membrane of Bruch may be absorbed, though it offers considerable resistance in common with all elastic membranes. When this has occurred leucocytes are enabled to pass through into the retina and vitreous.

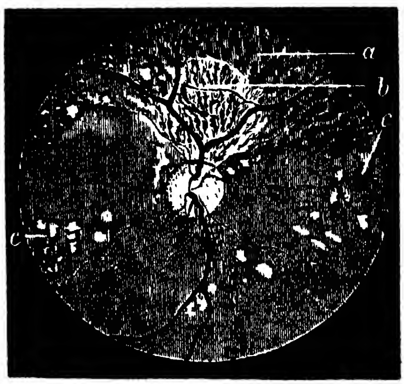


FIG. 190.—Atrophy after syphilitic choroiditis. (Nettleship, after Hutchinson.) *a*, atrophy of pigment epithelium; *b*, atrophy of epithelium and choriocapillaris, exposing the large choroidal vessels; *c*, spots of complete atrophy, many with pigment accumulation.

Owing to the fibroblastic activity of the choroidal stroma the exudates become organised, so that a small white mass of fibrous tissue is formed, which destroys the normal structures of the choroid and retina, and fuses the two membranes firmly together. The colour of the spots therefore gradually changes to white, partly due to the fibrous tissue deposited, partly to thinning and atrophy, whereby the white reflex from the sclerotic is permitted to shine through (Fig. 190).

The pigment of the retinal pigment epithelium is extremely

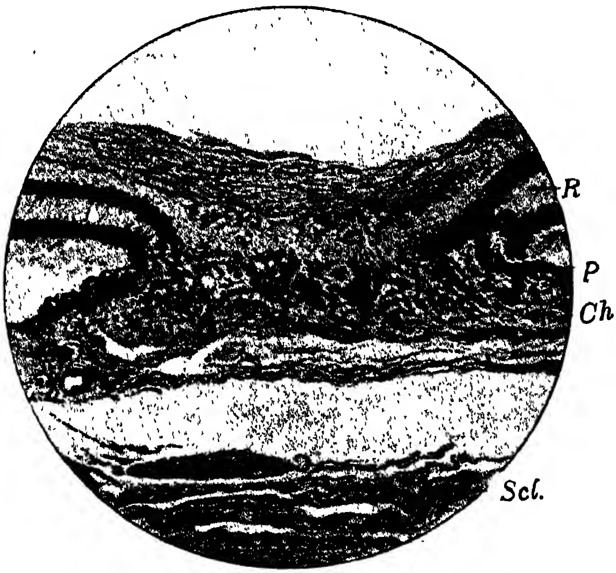


FIG. 191.—Section of a patch of disseminated choroiditis, showing the fusion of the degenerated retina (*R*) and choroid (*Ch*) (× 60). Note the heaping up of the retinal pigment epithelium (*P*) at the edges of the adhesion. *Scl.*, sclerotic.

resistant, even though the cells which contain it be destroyed. It tends to become heaped up into masses, partly intra-, partly extra-cellular: moreover the pigment cells are stimulated to proliferate. Isolated masses of black pigment are thus formed in the white areas, but more particularly at the edges, so that in the atrophic stage white spots surrounded by a black zone of pigment are seen (Fig. 191). The process has then reached its natural termination, and these spots remain permanently almost unaltered. They are much more sharply defined than the actively inflammatory spots.

Meanwhile fresh foci arise and pass through the same stages, until finally the whole fundus may be covered with atrophic spots. In the milder cases only a few spots are formed and the exudates in the vitreous become absorbed. In the more severe the spots are very numerous, the vitreous opacities increase, and finally the nutrition of the lens suffers and a complicated cataract (*q.v.*) results. Owing to the transiency of the acute stage the atrophic stage naturally comes much more frequently under observation.

The symptoms in the early stages are principally the defects of vision due to the retinal lesions and to cloudiness of the vitreous. The spots are slightly raised, so that the contour of the retina is altered. This causes distortion of the images, giving rise to similar appearance of distortion of the objects seen—metamorphopsia: thus straight lines appear to be wavy or bent in various directions. Frequently objects appear smaller than they are—micropsia; sometimes larger—macropsia: these results are due to separation or crowding together respectively of the rods and cones. They are not likely to be noticed unless the macular region is involved. Subjective symptoms of light—photopsiæ—occur, such as flashes of light, due to retinal irritability. These subjective symptoms are often accompanied by the perception of a black spot in front of the eye, corresponding with the lesion—*positive scotoma.*

In the later stages the affected spots are incapable of giving rise to visual impulses, so that *negative scotomata* exist in the field of vision, *i.e.*, though there is no perception of a spot in front of the eye there is a hiatus in the field of vision of the same nature as the normal blind spot. Their relative importance depends upon their situation. Peripheral scotomata may pass unnoticed, central scotoma destroys direct vision; in the latter case peripheral vision still permits the patient to get about well, but all fine work is impossible.

The disease is chronic, organisation of the exudates taking several weeks. The occurrence of fresh spots may extend the acute stage over months, and the ultimate defects are permanent. It is often bilateral.

The disease is usually due to syphilis, generally acquired, sometimes congenital, and hence associated frequently with interstitial keratitis; but in many cases the cause is obscure, sepsis, tuberculosis, anæmia and disorders of nutrition being assigned. The changes produced by myopia cause similar signs and symptoms; they are not inflammatory, but degenerative from the commencement (Chap. XXIV.).

Treatment is primarily that of the ætiological factor—syphilis or such cause as can be discovered. Iodide of potassium may assist absorption in all cases, and should be administered. The general régime advised for cyclitis (*q.v.*) is suitable in these cases. Marked irritative symptoms indicate the use of dark glasses, the abandonment of all near work, and sometimes the application of leeches.

Anterior Choroiditis is also usually syphilitic, and manifests itself in the same form as disseminated, but is confined to the peripheral parts of the fundus. On this account it is frequently discovered only by the ophthalmoscope. Similar changes are also sometimes found in high myopia. Simple pigmentary changes at the periphery occur in old people as a senile degeneration.

The periphery of the fundus is often peppered over with minute spots of pigment in congenital syphilitics; this is possibly a purely retinal affection. It can only be distinguished by degree from a similar pigmentation which may be a mere idiosyncrasy.

Central Choroiditis occurs in disseminated choroiditis, and in certain rare forms. In Förster's areolar central choroiditis the spots are said to behave in exactly the opposite manner to those of disseminated; they are first black, then enlarge, becoming white in the centre, and finally quite white. The disease extends outwards, the peripheral spots being always the most recent.

Juxtapapillary Choroiditis (*Retinochoroiditis juxtapapillaris*) (Jensen) occurs in young persons, as an exudation close to the disc, oval in shape and about the same size as the disc. The exudates cover the retinal vessels, and there are vitreous opacities and sometimes "k.p." There is a sector-shaped defect in the field of vision. The cause is unknown. The inflammation slowly subsides, leaving a patch of atrophy. Recurrence may take place.

Diffuse Choroiditis is characterised in the early acute stage by one or more plaques of yellowish white or grey areas, shading off at the edges into normal fundus. The patches spread and coalesce so that the greater part of the fundus may be finally involved. The exudates gradually organise, leaving white areas in which the larger choroidal vessels persist, as a characteristic band-like network. The retinal pigment becomes heaped up into dense black, irregular spots, variously grouped. The retinal vessels course over the patches little changed in appearance. The coalesced areas leave islands and spaces of

normal fundus between them when organisation is complete. Fresh exudates occur simultaneously with the organisation of older ones, and sometimes the edge of a patch appears to creep over the fundus like the advance of a myxomycetes.

Some of these cases are syphilitic, some tuberculous (*q.v.*). In others the cause cannot be traced, but probably many are due to metastatic bacterial invasion (*vide infra*, Metastatic Endophthalmitis).

Purulent Choroiditis. See p. 462.

Tubercle of the Choroid occurs in acute or miliary and chronic forms. Miliary tubercles are found in the late stages of acute

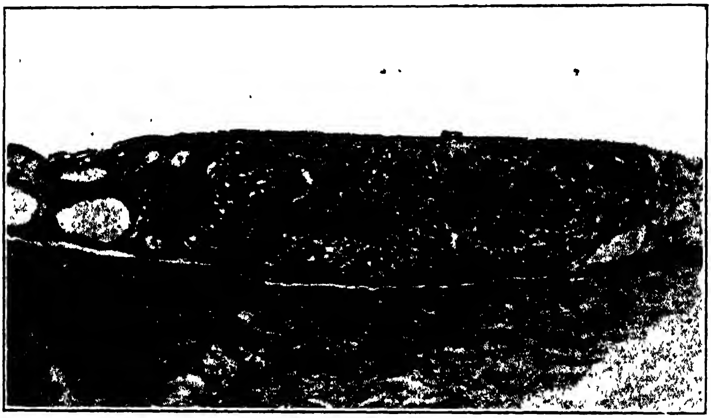


FIG. 192.—Section of miliary tubercle of the choroid (× 60), from a child with hip disease who died of meningitis. Note the giant cells and the small round cells at the periphery.

miliary tuberculosis, especially tuberculous meningitis. Occasionally they may be seen before there is any evidence of meningitis or generalised tubercle. They are very common in the late stages of tuberculous meningitis in children, though they may appear only a day or two before death. My own observations, clinical and post-mortem, lead me to think that they are nearly always present in this disease. Ophthalmoscopically they appear as round, pale yellow spots, most frequently observed in the neighbourhood of the disc, though any part of the choroid may be attacked. Generally only three or four spots are seen, but as many as sixty or seventy have been found. They vary in size from pin-point specks to 1 or 2 mm. in diameter. They usually project slightly, so as to

raise the retina, but the inner surface is often quite flat, while the outer surface projects into the sclera. They afford most important diagnostic evidence of tubercle in cases of meningitis and obscure general disease. Microscopically they consist of typical giant-cell systems, containing a variable number of tubercle bacilli (Fig. 192).

Chronic tubercle may occur as a diffuse or disseminated inflammation affecting large areas or the whole choroid, and characterised by the extensive development of granulation tissue; or, more rarely, as a solitary or conglomerate mass, simulating sarcoma, but usually showing definite signs of inflammation, *e.g.*, œdema of the retina, vitreous opacities, &c. The primary systemic focus is rarely, if ever, in the acute stage. The diffuse form shows ophthalmoscopically areas raised somewhat above the surrounding fundus, covered by œdematous retina, and with hazy edges. There are usually vitreous opacities. The mass consists of granulation tissue containing giant cells, spreads until it involves the retina and may finally fill the posterior part of the globe. Similar ophthalmoscopic appearances are met with resulting from metastatic choroiditis (*q.v.*), and from changes following localised hæmorrhage into the deep layers of the retina (so-called "massive exudation" in the retina (*vide* p. 365)). von Pirquet's test or injection of tuberculin may afford help in the diagnosis. Such cases were formerly all diagnosed as tubercle of choroid, but this is certainly not the case. They occur both in children and young adults, and may subside, particularly in the latter, leaving large areas of choroido-retinal atrophy. Possibly the cases which behave thus are not really tuberculous.

Certainly true tubercle of the choroid may extend indefinitely, especially in children, and the conglomerate type usually does so. The sclerotic becomes involved; perforation takes place, usually near a vortex vein or an anterior perforating ciliary vessel, a fungating mass appearing under the conjunctiva. Both types in very young children may nearly simulate retino-blastoma (especially "glioma endophytum" (*q.v.*)), constituting one form of pseudoglioma (*q.v.*).

Treatment. No local treatment is indicated in miliary tubercle of the choroid. The patient quickly succumbs to the general disease. In diffuse and conglomerate tubercle treatment with tuberculin should be instituted in the early stages, but if the eye is extensively involved it is best to enucleate it, and thus remove a dangerous focus from which the organism

may be disseminated into the system. Complete rest (even sanitorium treatment) is essential, and should be combined with sunlight or ultra-violet treatment. Diathermy, as used in detachment of the retina (*q.v.*), has been used for conglomerate tubercle (Weve).

Brucellosis. Brucellosis may cause a chronic choroiditis indistinguishable from other types of diffuse choroiditis. For diagnosis and treatment see p. 268.

Metastatic Endophthalmitis. Endogenous bacterial infection of the eye manifests itself in various ways. Most commonly the uveal tract is affected, either as a whole (metastatic uveitis) or in its individual parts, as for example in gonorrhœal iritis. The milder forms of iridocyclitis (*q.v.*) and uveitis are probably due to toxins circulating in the blood-stream, and derived from bacterial foci in other parts of the body, *e.g.*, the mouth, the generative apparatus, especially in women, the intestinal tract and so on. It is certain, however, from anatomical examination, that actual bacterial embolism occurs, and in these cases two facts stand out prominently. First, various organisms show a special selectivity for the various structures of the eye. Thus the tubercle bacillus never attacks the retina primarily, though this structure is often involved secondarily in tuberculous disease of the choroid. Second, except in the case of extremely virulent organisms, such as the streptococcus, the inflammation set up by endogenous infection is usually less severe than when the organism is introduced directly into the eye from without. Thus, if the eye is infected with pneumococci by a perforating wound an exogenous panophthalmitis (*q.v.*) is likely to be set up, leading to the complete destruction of the organ. So much is this the case that saprophytic organisms, such as the bacillus subtilis, which are non-pathogenic in other parts of the body, may cause panophthalmitis. If, however, an organism such as the pneumococcus invades the eye by way of the blood-stream, though an intense inflammation results it tends to subside more rapidly than in the exogenous cases. Probably the organism becomes attenuated in the blood-stream and tissues through the controlling effect of specific anti-bodies. In this manner virulent bacteria may set up endophthalmitis of every grade of severity. In the days when puerperal fever was prevalent, intense metastatic panophthalmitis, often attacking both eyes, was not uncommon. It was characterised by the appearance of an hypopyon and the rapid development of a yellow pupillary reflex due to pus in the vitreous. An in-

tense metastatic uveitis, involving iris, ciliary body and choroid, and due to bacterial invasion, was set up. Such cases progressed exactly like ordinary exogenous panophthalmitis (*q.v.*).

They are now seldom seen, but cases of metastatic bacterial invasion often of obscure origin still occur in which the early stage of uveitis with hypopyon is present. They occur in the course of infectious diseases, especially pneumonia, influenza, measles and scarlet fever, and of meningitis, dysentery, &c.

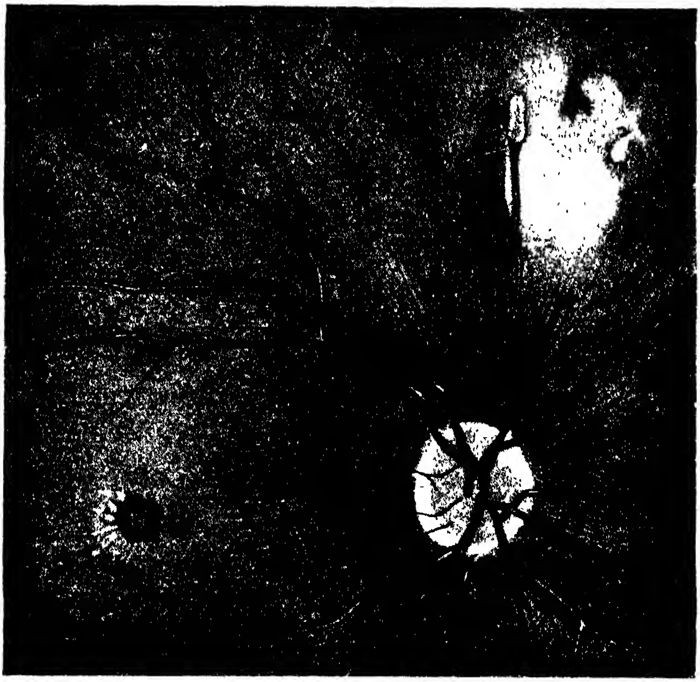


FIG. 193.—Metastatic choroiditis.

Ophthalmoscopically the media are hazy, so that the yellow œdematous retina is only dimly seen. Under treatment the condition gradually subsides, not infrequently with the restoration of useful vision. In more severe cases the inflammation causes destruction of the ciliary processes, the intraocular tension falls, and the eye gradually shrinks. In children the inflammation is probably often due to the diplococcus intracellularis of Weichselbaum and is followed by an exuberant development of new fibrous tissue of cyclitic origin. These

cases are not easily distinguished clinically from glioma, and form the largest group of so-called pseudoglioma (*q.v.*). In the milder cases there is less development of cyclitic fibrous tissue in the vitreous and less scarring of the retina, but whitish atrophic spots and areas accompanied by pigment disturbance persist as evidence of the previous inflammation. Yet milder cases give rise to ophthalmoscopic appearances which may be mistaken for diffuse or conglomerate tuberculous diseases of the choroid (*q.v.*) (Fig. 193), or thrombosis of a branch of the central vein of the retina. In some cases the optic papilla has been the chief local focus, and an appearance of intense papillitis with much exudation extending into the neighbouring retina is seen. Some of the cases have been associated with furunculosis, and there is little doubt that they are due to metastatic infection with staphylococci. Others have been proved to be due to the pneumococcus, meningococcus, &c. Other obscure cases of retinitis with white spots or oval areas, usually near the disc, with or without hæmorrhages, are probably due to the same cause.

The presence of precipitates ("k.p.") on the back of the cornea and inconspicuous posterior synechiæ shows that in many cases of apparently localised endogenous choroiditis the whole uveal tract is really involved (uveitis).

The *treatment* depends upon the severity of the attack, and the possibility of determining the primary focus and the specific organism. In the worst cases the patient should remain in bed, and purgatives and the drugs for the relief of pain should be administered. Hot applications should be made and atropine instilled. Sulphonamide treatment should be tried (*vide* p. 695). If the eye becomes full of pus it should be enucleated or eviscerated according to the principles which govern the treatment of panophthalmitis of exogenous origin (*q.v.*).

In the milder cases purgatives and general tonic treatment are indicated. Atropine should be instilled to keep the eye at rest, and dark glasses worn. Counter-irritation with leeches or blisters to the temple may be indicated. If the primary source of infection can be discovered it must be treated radically. If the organism can be isolated vaccine treatment is indicated, and in the more obscure cases treatment with a polyvalent vaccine may be of some avail.

Suppurative or Purulent Choroiditis. See Metastatic Endophthalmitis (p. 343) and Panophthalmitis (p. 462).

Degenerative Changes may be post-inflammatory or primary. The former, culminating in localised spots of complete atrophy,

have already been considered. Generalised atrophy, more or less complete, is found in the later stages of glaucoma. The loss of nourishment to the retina causes atrophy of the outer layers and of the nerve fibre layer in these cases. Degenerative changes in the choroid often cause migration of pigment from the pigment epithelium into the more superficial parts of the retina. The pigment tends to become deposited in the perivascular lymph spaces of the veins, so that the retinal veins may be mapped out here and there by pigment. More noticeable ophthalmoscopically are jet-black branched spots of pigment, resembling bone corpuscles, and standing out in sharp relief. This condition is seen in its most typical form in retinitis pigmentosa (*q.v.*). An almost identical picture, though

FIG. 194.—Section of "colloid bodies," seated upon the membrane of Bruch. They are undergoing calcification, as shown by the deeper staining in the inner parts. They are covered with stretched pigment epithelial cells.

usually without the characteristic distribution of the pigmented spots, may result from choroidal atrophy due to other causes, *e.g.*, syphilis.

Primary choroidal degeneration may be localised or general: localised choroidal atrophy, apart from the post-inflammatory forms, is usually either central or circumpapillary.

Central Choroiditis, or more properly *central choroidal atrophy*, is most commonly the result of myopia (*q.v.*), syphilis, contusion (Chap. XXI.), or old age.

Senile central choroidal atrophy assumes two chief forms. In *central guttate choroïditis* (Tay's choroiditis) there are numerous minute yellowish white spots in the macular region (Plate X. Fig. 1). They may increase in numbers, but otherwise they remain stationary. They are always small, usually round, but the larger spots may have crenated edges, thus showing signs of fusion. There may be indefinite signs of greyish

PLATE X.

FIG. 1.—"Tay's choroiditis.

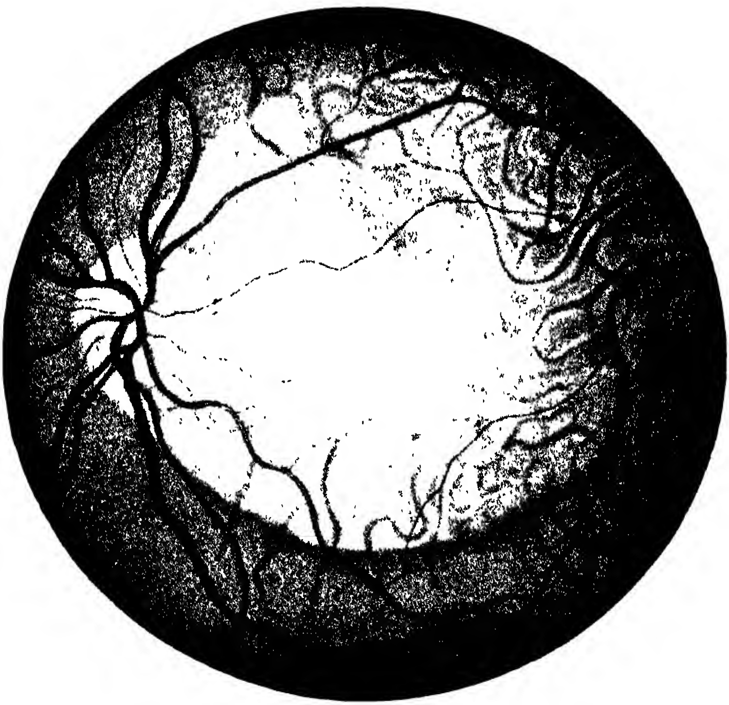


FIG. 2.—Central senile areolar choroidal atrophy.

[To face p. 346.

PLATE XI.

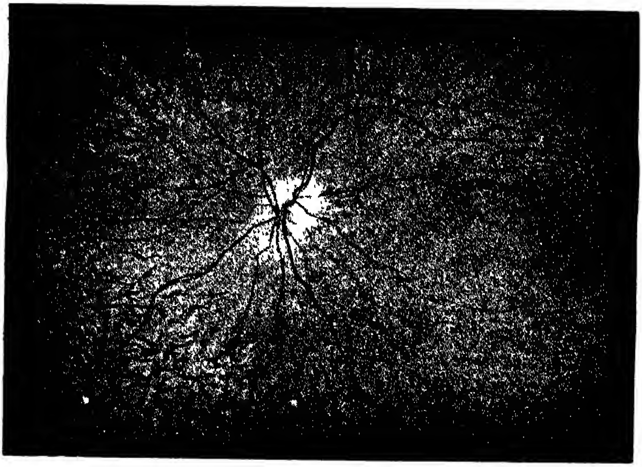


Fig. 1.—Retinitis pigmentosa.

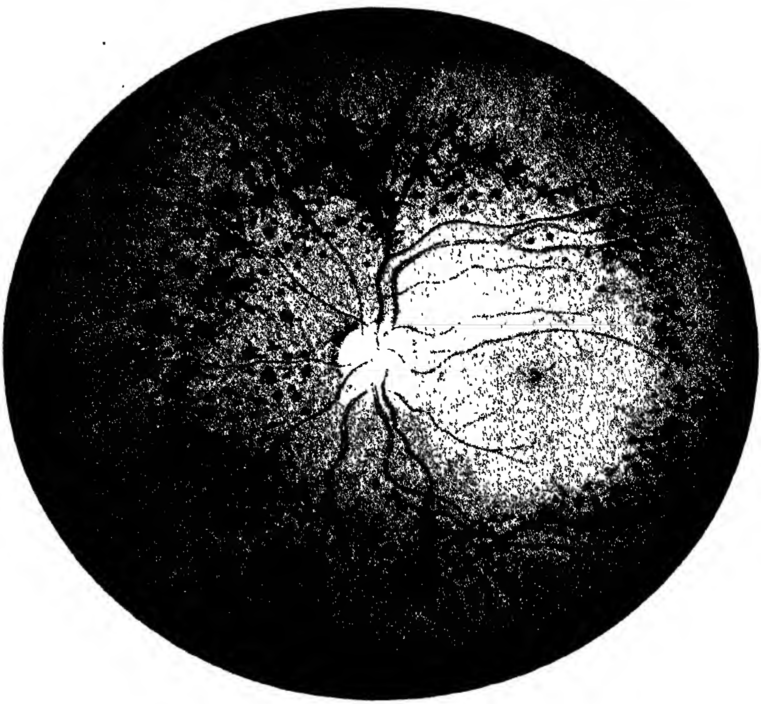


Fig. 2.—Retinitis pigmentosa.

pigmentation of the edges of the spots, due to the fact that the pigment epithelium is stretched over them (Fig. 194). The condition is bilateral. They have been mistaken for diabetic or albuminuric retinitis (*q.v.*) and retinitis punctata albescens (*q.v.*). They are easily distinguished from the former, in which the spots are much brighter and more glistening, and are often arranged in the form of a fan or star; and from the latter, in which the spots are not limited to the macular region and are much whiter. In neither case is there any obvious disturbance of pigment. Central guttate choroiditis causes *per se* little impairment of vision, but other senile changes are often present and account for defective sight. The spots are due to peculiar hyaline excrescences on the surface of the choroid, commonly known as *colloid bodies* (Fig. 194). They are of the same nature as Bruch's membrane, and like it are secreted by the pigmented epithelial cells.

Central areolar choroidal atrophy—to be carefully distinguished from Förster's areolar choroiditis (*vide* p. 340)—appears as a large circular or oval patch of degeneration in the macular region in which the choroidal vessels are visible, owing to atrophy of the retinal pigment epithelium (Plate X., Fig. 2). As a result of atrophy of the choroid the sclerotic shines through and the patch is white, though traversed by choroidal vessels. Only the larger choroidal vessels are seen, the smaller ones having disappeared, and even the large ones appear smaller than usual owing to degeneration of the walls. There is an absolute central scotoma. The condition is to be feared in cases of cataract in which perception of light seems defective; hence the great importance of investigating the fundus thoroughly in cases of immature cataract. It is possible that this form of central choroidal atrophy is due to extravasation of exudates.

Besides these conditions, and much commoner, minute changes limited to the area at and immediately around the fovea centralis occur not infrequently in old people and lead to grave disturbance or abolition of central vision (*central senile macular degeneration*). It is generally necessary to dilate the pupil with cocaine or homatropine in order that they may be seen, care being taken to instil eserine when the examination is finished (*vide* p. 311). When central vision is very poor in an elderly patient and no cause can be found to account for the defect, such as error of refraction, tobacco amblyopia, cataract, glaucoma, &c., pathological changes will probably be found at the macula. In the early stage the fovea is surrounded by

a ring of very fine pigment spots. The stippling is more sharply defined on the foveal side, which usually has a circular or crenated edge ; it diminishes rapidly peripherally, where the fundus becomes normal. The fovea gradually becomes paler in colour and the stippling denser, the change being associated with increasing failure of vision, until eventually the small central scotoma becomes absolute. The progress is usually slow. Both eyes are affected, but one is usually attacked before the other, and many months may intervene. It is often attributed on slender evidence to septic absorption. In the majority of cases, however, it is probably due to an obliterating sclerosis of the small vessels in the subjacent chorio-capillaris.

Sometimes a round white or yellowish patch, about the size of the disc, is seen at the macula and may be bilateral. Occasionally it appears as a raised mass of fibrous tissue (*Disciform degeneration at the macula*). The patient is usually old with obvious disease of the retinal vessels. Some of these patches undoubtedly follow hæmorrhages.

Treatment. The treatment of these degenerative changes is very unsatisfactory. When central senile changes are seen early the treatment of any source of sepsis, *e.g.*, pyorrhœa alveolaris, should be carried out. Strychnine, thyroid extract and general tonic treatment is usually applied, but without much success. Smoking should be prohibited (*vide* p. 397). The patient's mind may be relieved to the extent that the rest of the field of vision is not likely to become affected, so that, although unable to read or do fine work, he can get about freely. In the early stages reading is facilitated by the use of a black mask in contact with the paper and exposing only one line of print ; or by the use of a magnifying glass, especially a " reading crystal," *i.e.*, a plano-convex lens in contact with the paper.

Circumpapillary Choroidal Atrophy is found in myopia (*q.v.*) and in late stages of glaucoma.

Detachment of the Choroid. The choroid is often found detached from the sclerotic in eyes which have been lost by plastic iridocyclitis, glaucoma, &c. Detachment may also occur from severe hæmorrhage or new growth. These cases cannot be diagnosed clinically. Fuchs has shown that the condition is not uncommon after iridectomy for glaucoma and cataract extraction, and attributes it to slight separation of the ciliary body, so that the aqueous percolates from the anterior chamber into the suprachoroidal space. It occurs

during the first days after the operation. The anterior chamber is shallow or abolished, and on ophthalmoscopic examination the detached choroid is seen as a dark mass behind the lens. It may be visible as a dark brown mass by oblique illumination. Detachment of the choroid occurs not infrequently after trephining for glaucoma. The prognosis is usually good, the choroid becoming replaced and the anterior chamber re-established.

Sarcoma of the Choroid. See p. 424.

Primary Affections of the Retina

Primary affections of the retina in their most severe manifestations are almost always the result of some general disease, and should, therefore, be properly regarded as symptomatic diseases to which the term "retinopathy" is sometimes more applicable than the classical term "retinitis." The metabolic changes in the retina are so rapid that it seems almost immune from fatigue in the proper sense of the word; hence retinal fatigue plays little or no part in causing asthenopia (*vide* p. 531). *Hyperæmia* of the retina is often described, but can never be diagnosed with certainty in the absence of definite pathological signs in the retinal vessels (*vide infra*).

These affections in general give rise to the following symptoms and signs, only some of which need be present in individual cases. There is usually some change in visual acuity. Rarely it is increased in the early stages; more commonly it is diminished throughout. There may be concentric diminution of the field of vision, or scotomata may be present corresponding with the areas specially affected. There may be metamorphopsia, micropsia, or macropsia (*vide* p. 339). The light sense is diminished, and photophobia may be present. Pain is almost invariably absent, though discomfort may be experienced.

The ophthalmoscopic signs may be diffuse or localised. There may be general œdema, manifesting itself as a faint, diffuse haze, obscuring details, so that the normal bright red appearance is replaced by a paler cloudiness, often with definite white streaks, especially along the course of the vessels; or there may be circumscribed areas of exudation. The latter appear as white spots, discrete or confluent, or yellowish plaques, varying in size. They are not pigmented and the edges are ill-defined, so that there is little danger of mistaking them for patches of choroidal atrophy. The blood vessels usually show marked changes. The veins are

distended, often irregular, darker than normal, and tortuous; the arteries are less altered, but the finer branches are also tortuous. Hæmorrhages are common, though they are not in themselves evidence of primary retinitis. When occurring in the superficial layers they are flame-shaped, with feathery edges, situated particularly along the course of the vessels; when in the deeper layers they are round, with better-defined contours.

The optic nerve, being in anatomical and physiological continuity with the retina, often suffers with it; when this occurs to a marked extent the condition is called *neuro-retinitis*. The margin of the disc is then obscured and often shows a radial striation. The disc is red, and may be measurably swollen, though seldom, if ever, so much as in the condition known as choked disc.

Retinal atrophy, which follows severe retinitis, is shown by permanent whitish or yellow opacity, with diminution in the size of the vessels, which are often bordered by white lines. The optic disc may show all the signs of advanced atrophy (*vide* p. 401).

It has already been pointed out that retinitis is most frequently the symptom of an internal disease. It is therefore usually bilateral. The diseases most commonly causing the condition are syphilis, vascular disease, nephritis, diabetes, and leucæmia.

The *treatment* of retinitis consists in giving the eyes complete rest and in combating the general disease which is the cause. All near work is forbidden and the eyes are protected with smoked glass, or even confinement in a darkened room. Whether due to syphilis or not, mercury and iodides are given with a view to aiding the absorption of exudates and restoring the transparency of the vitreous, if this is affected. Mercury is contra-indicated in renal retinitis. These means are supplemented by purgatives, diaphoretics, and tonics.

The blood vessels of the retina are peculiarly subject to disease, partly as a factor in the inflammation, but more prominently as a concomitant of general disturbance or disease. It will be well, therefore, to discuss the commoner forms of vascular disease of the retina before passing on to describe the chief types of retinitis in greater detail.

Vascular Disorders of the Retina

Anæmia may be part of general anæmia or due to local causes. It may be sudden or slow in onset. Sudden anæmia is seen in embolism of the central artery of the retina (*q.v.*)

and in quinine amblyopia (*q.v.*). Ophthalmoscopically there is great attenuation of the retinal vessels and the optic disc is pale. Spasmodic constriction of the retinal arteries has been described in migraine (*vide* p. 416), but it is doubtful if it occurs. I have seen one case of spasm of the retinal arteries in one eye, giving rise to the symptoms of embolism of the retinal arteries: it passed off during the actual ophthalmoscopic examination. The retinal vessels constrict under high oxygen concentration in the blood, and dilate in anoxæmia. Anæmia of slow onset is seen in atrophy of the retina from

FIG. 195.—" Copper-wire " arteries, degeneration of the walls of a vein, and white spots of degeneration.

any cause, such as previous retinitis, and in disease of the vessel walls as part of a general vasculitis. In both cases the vessels are attenuated, and sometimes in the former, and commonly in the latter, the walls become thickened and visible as white lines bordering the red blood stream; eventually the vessels may be transformed into white strands or may even disappear.

Embolism of the Central Artery of the Retina causes sudden and complete retinal anæmia. The retina in rabbits dies within half an hour after complete blockage of the central artery, but probably survives longer in man. The eye, usually the left, becomes suddenly quite blind. Examination of the fundus

reveals a very typical picture (Plate XII., Fig. 1). The larger arteries are reduced to threads, the smaller are invisible. The veins are little altered except on the disc, where they are contracted. Within a few hours the retina loses its transparency, becoming opaque milky-white, especially in the neighbourhood of the disc and macula. Owing to the opacity of the retina the outlines of the disc, which is abnormally pale, are obscured. At the fovea centralis, where the retina is ex-

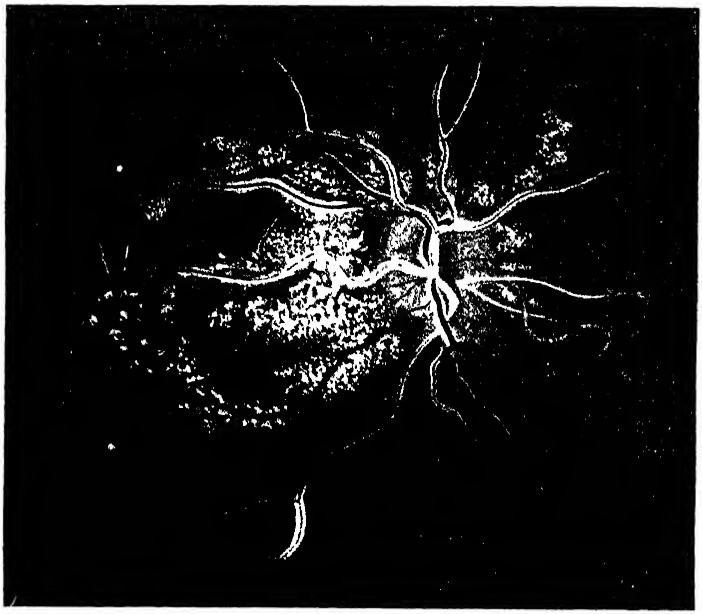


FIG. 196.—Perivascular changes in the retinal vessels.

tremely thin, the red reflex from the choroid is visible. It appears as a round "cherry-red" spot, presenting a strong contrast to the cloudy white background. The peculiar tint of the spot is due to this contrast. In the majority of cases there is no hæmorrhage, as was once thought, though hæmorrhages here and in the immediate neighbourhood do occur rarely. The contrast sometimes brings into relief minute blood vessels near the macula which are otherwise invisible.

Sometimes the obstruction to the blood flow is not complete, or the flow may be partially restored in the course of a few days. Another peculiar phenomenon may be observed or may be

induced by gentle pressure upon the globe. In some of the vessels, usually veins, the column of blood is broken up into red beads separated by clear interspaces. The beads move in a jerky fashion through the vessels, sometimes in the normal direction of blood flow, sometimes in the opposite direction. If the veins are easily emptied of blood or arterial pulsation is

FIG. 197.—Obstruction of the Central Artery (Coats). Showing central vessels posterior to lamina cribrosa. The artery, to the left, is collapsed and obliterated by proliferation of endothelium. The vein is concentrically narrowed by thickening of its walls, not by endothelial proliferation.

produced by slight pressure on the eye-ball, it is evidence of incomplete blockage.

The retinal œdema, or more probably coagulation necrosis, takes several weeks to clear up. The membrane regains its transparency, but is completely atrophic. The vessels are contracted or reduced to white threads. The disc is atrophic. If there have been hæmorrhages spots of degeneration replace them, and cholesterin crystals and pigmented spots may be seen in the papillo-macular region.

In some cases a certain degree of central vision persists in spite of apparent complete occlusion of the central artery. It is due to the presence of cilio-retinal arteries (*vide* p. 125),

which, when present, always supply the macular region, and naturally escape occlusion; or to a macular branch of the central artery given off proximal to the block. The remainder of the field of vision is lost. In rare cases a cilio-retinal artery alone becomes blocked.

After the first stage, in which the arteries are threadlike from sudden partial or complete arrest of blood flow, the vessels refill slightly, showing a small stream in more or less normal arteries. This is due to establishment of a feeble collateral circulation through the anastomoses with the ciliary

FIG. 198.—Retinal vein with enormously thickened wall and narrow lumen (Coats). The perivascular lymph space is dilated.

system round the disc (*vide* p. 11). At a later stage the vessel walls become thickened, so that the thin red line is bordered by white lines, the last stage of threadlike sclerosed arteries. The final stage of complete obstruction of the central artery shows a pale atrophic disc due to degeneration of the nerve fibres of the retina and disappearance of the capillaries of the nerve head, threadlike retinal arteries containing blood only on and near the disc, and larger veins containing blood: the rest of the fundus is of normal colour and appearance. Unusual freedom of collateral circulation may account for the considerable recovery of vision which occurs in rare instances, almost

PLATE XII.

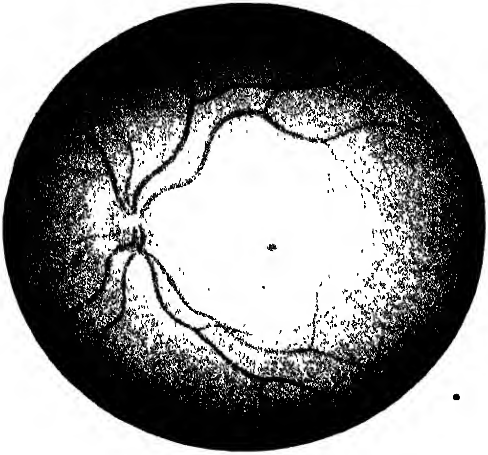


FIG. 1.—Embolism of the central artery

FIG. 2—Thrombosis of the central vein.

[To face p. 354.

PLATE XIII.

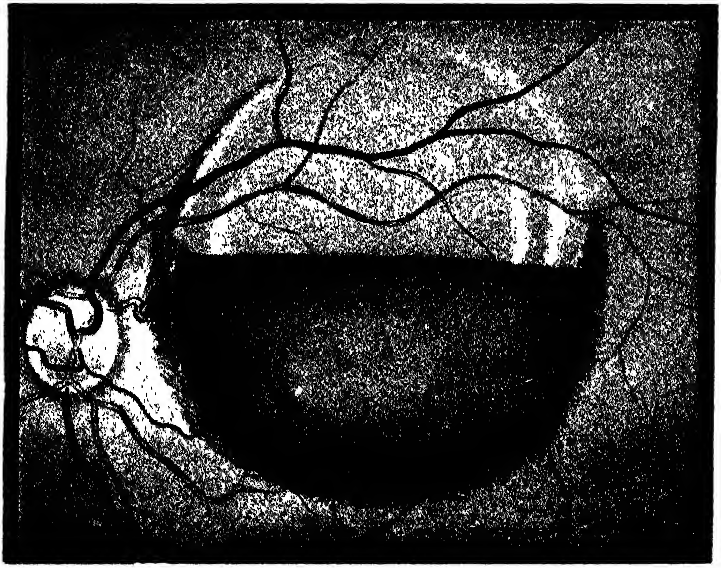


Fig. 1.—Subhyaloid hæmorrhage.

Fig. 2.—Retinitis proliferans.

invariably limited to the temporal field. In rare cases, too, visible anastomoses have been seen on and at the edge of the disc.

It might be thought that the intraocular pressure would force the blood out of the veins; but there is normally some obstruction to the outflow at the lamina cribrosa, where the venous pressure is lowest. This is shown by the occurrence of venous pulsation (*vide* p. 126). The blood is dammed back by the constriction of the elastic fibres of the lamina cribrosa.

In other cases the embolus is arrested in a branch of the central artery. The area supplied by this branch is then affected alone. In the early stages the corresponding scotoma is usually somewhat indefinite, but later it settles down to a permanent sector-shaped defect.

When perception of light is lost the pupil is large and the direct reaction to light fails. The intraocular tension is normal, as might be anticipated.

There is occasionally the history of prodromal attacks of temporary obscuration of vision. Some are due to local arterial disease associated with temporary diminished blood pressure, and arterial spasm may play a part.

Embolism of the central artery, like cerebral embolism, occurs with mitral stenosis, especially if there has been fresh endocarditis. This factor is, however, very frequently absent, and it is probable that most cases are really due to thrombosis. There has been endarteritis, due to general arteriosclerosis from nephritis, &c., and the already narrowed lumen of the vessels has become suddenly occluded. The onset may be less rapid in such cases and preceded by premonitory symptoms, such as obscuration of vision, &c. Other cases render it probable that the condition may be due simply to spasm of the walls of the artery, thus accounting for some remarkable cures. In others it may be that an embolus has been forced on into a smaller, more peripheral branch.

The condition has been observed at ages varying from fifteen to eighty. Rare cases of simultaneous bilateral blocking of the central arteries have been recorded.

The blockage, whether due to embolus or thrombosis, is nearly always at the lamina cribrosa, where the vessels normally become slightly narrowed (Fig. 197). The retina undergoes atrophy of the nerve-fibre and ganglion cell layers, with preservation of the outer layers, which receive their nourishment from the chorio-capillaris of the choroid.

Treatment is seldom of any avail, but attempts should be made to drive the obstruction on into a less important branch if the case is seen early. Massage of the globe, and paracentesis have been employed for this purpose ; such measures must be adopted without delay. Inhalation of amyl nitrite is useless—as might be expected—since it lowers the general blood pressure and leads to passive constriction of the intraocular vessels. A definite case of cure of embolism of a branch of the central artery by the subconjunctival injection of acetylcholine has been reported (Orr and Young): 8 minims (B.D.H.) should be injected into Tenon's capsule and behind the equator. The drug causes great dilatation of both arteries and veins in the retina, the embolus being driven on into a smaller and more periphéral branch.

Amaurotic Family Idiocy (*Syn.—Tay-Sachs Disease*) shows ophthalmoscopic signs resembling those of embolism of the central artery, but of quite different origin. The disease occurs almost if not quite invariably in Jewish children, and commences during the first year of life. Several members of a family may be affected. The apparently healthy child becomes gradually blind, with muscular weakness and wasting, and mental apathy passing into idiocy. Death follows in from one to two years. The opthalmoscopic picture is very characteristic and the same in every case. There is a round brilliantly white area at the macula, fading off peripherally into the normal fundus. In the centre of the patch is a brownish-red, circular spot at the fovea. In the later stages there is optic atrophy. It is always bilateral. The disease is a primary lipoid neuronic degeneration of the whole of the central nervous system, including the ganglion cells of the retina, associated with profuse overgrowth of neuroglia.

Maculo-cerebral Family Degeneration has some points of resemblance to amaurotic family idiocy, and has been regarded as a delayed or juvenile form of the disease, but should be carefully distinguished from it. It is a familial disease, occurring in other than Jewish children, and commencing at a later age, usually at about six or eight years. It is relatively commoner in Sweden (Sjögren). Defective vision, with central scotoma, is accompanied by weak intellect, convulsions and spasticity. Ophthalmoscopically the discs are pale and the vessels small. At the macula there are yellowish-grey spots and granular pigmentation, and there may be pigmentation in other parts of the retina. The ophthalmoscopic picture varies much in different cases. Similar macular degeneration beginning between the ages of twelve and fourteen has been

seen as a familial disease without cerebral deterioration (*Stargardt's Disease*).

Degenerative Changes in the Retinal Vessels, apart from their interest as a local manifestation of disease, are of the utmost importance in general prognosis. They may be the first evidence of arteriosclerosis, and particularly of disease of the cerebral vessels, pointing to the danger of cerebral hæmorrhage, and indicating lines of treatment which may prolong life. Disease of the retinal vessels is almost invariably associated with disease of the cerebral vessels, but disease of the latter may be present when there are no ophthalmoscopic signs of disease of the retinal vessels (Foster Moore). Undue tor-

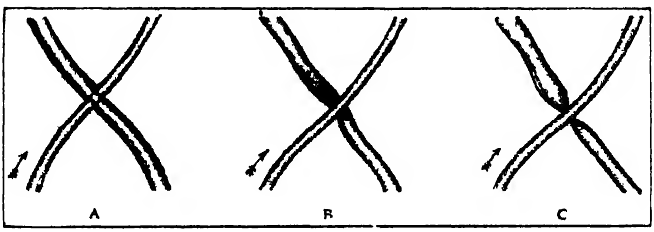


FIG. 199.—Retinal vein crossed by an artery (Marcus Gunn). A. In health, the underlying vein dimly traceable beneath the artery. B. In early stage of arteriosclerosis, the vein somewhat displaced in the direction of the arterial circulation, and its blood flow slightly obstructed. C. In advanced stage of arteriosclerosis, the vein greatly narrowed where crossed and distended on the peripheral side.

tuosity of the vessels is of no significance unless accompanied by other abnormalities, such as irregularity in the size and breadth of the arteries, so that stretches of the vessel are much constricted, alternating with normal or somewhat dilated portions. These changes in the lumen are due to endothelial proliferation in the intima. Minute miliary aneurysms are seen in rare cases. The normal light reflex from the vessel walls is often unusually bright and broad, especially in vessels at some distance from the disc ("copper-wire" arteries) (Fig. 195). Under normal conditions it is possible to see a vein through an artery at a point of crossing; in arteriosclerosis the artery loses its translucency so that the vein is obscured. Moreover, the artery exerts an abnormal pressure on the veins so that the blood flow is obstructed; the vein seems therefore to stop at

the crossing and is more distended on the distal side than on the side towards the disc (Fig. 199). Sometimes the vein appears to be pushed aside by the crossing artery; in severe cases the vein, whether crossing above or below the artery, is diverted so that it crosses at right angles, the shortest possible route. The veins may also exhibit a beaded appearance, with alternate constrictions and dilatations. More pronounced changes make the walls of the vessels visible, so that the blood column, often narrowed, is bounded by white lines, the thickened fibrous walls (Fig. 196); this may affect both arteries and veins, usually only individual vessels, in a portion of their course (Figs. 195, 196).

The changes indicated lead to increased permeability of the walls, and increased internal pressure, due to general disease, supplements this defect, and causes undue extravasation of lymph, and even hæmorrhage. Œdema of the retina thus arises, manifesting itself as a greyish opacity around the disc, or in spots along the course of the vessels. Hæmorrhages occur as linear striated extravasations along the vessels, or as round spots scattered over the fundus.

These changes occur most frequently in elderly people and are seldom entirely absent in the aged. They are specially pronounced in cases of chronic nephritis, syphilis, some forms of poisoning, especially by lead and probably by auto-intoxication, notably that of intestinal origin. They are frequently associated with high blood pressure, and always indicate the necessity for exhaustive examination of the circulatory and excretory systems. On the other hand, the blood pressure may be normal, possibly owing to cardiac dilatation, and these cases are probably more subject to thrombosis (Foster Moore). The prognosis as regards life in retinal vascular disease is decidedly better than in cases of renal retinitis (*q.v.*), though the patients may die suddenly from cerebral hæmorrhage or thrombosis. Vascular changes are more frequent in women than men, though the former seem to be more tolerant of high blood pressure than the latter (Foster Moore). They may be very marked and extensive in quite young people as the result of congenital syphilis; rarely as an hereditary condition without syphilitic taint. Extensive disease of the retinal vessels, with much diminution in their calibre, so interferes with the nutrition of the retina that consecutive atrophy of the optic nerve not infrequently follows. The ophthalmoscopic appearances of the vessels and disc then closely resemble those found in advanced cases of retinitis pigmentosa. This condition may

be accompanied by groups of sharply defined small white spots in the retina and even a fan- or star-shaped figure at the macula (Foster Moore) (*arteriosclerotic retinitis*). It is frequently unilateral, thus differing from renal retinitis.

Angioid Streaks. Dark brown or pigmented streaks, which anastomose with each other and resemble blood vessels in distribution, are sometimes seen ophthalmoscopically in retinæ which are undergoing degenerative changes. They differ in distribution from any normal set of vessels, are usually situated near the disc, at a deeper level than the retinal vessels, and are very irregular in contour. They are often associated with elastic pseudoxanthoma of the skin. They may be due to proliferation of new vessels into scar tissues (W. T. Lister), or to pigmented fibrous bands in the inner layers of the choroid (Verhoeff); but are probably due to changes in the elastic tissue of Bruch's membrane.

Hyperæmia may be arterial or venous. Arterial hyperæmia, characterised by fulness and tortuosity of the arteries, accompanies not only inflammation of the retina, but also inflammation of neighbouring structures, especially the uveal tract. Venous hyperæmia, characterised by dilatation and great tortuosity of the veins, is the result of impeded return of blood to the heart. It may be due to general venous congestion, seen in its most extreme form in congenital malformation of the heart (*cyanosis retinæ*), or to local causes. The latter most commonly affect the veins in the porus opticus, as is seen in moderate degree in glaucoma and optic neuritis, and in extreme form in thrombosis of the central vein of the retina. Increased intraorbital pressure, as from a tumour, may also impede the exit of blood from the eye. The veins are much enlarged and dark in colour in polycythæmia.

Hæmorrhages from the retinal vessels may be pre-retinal or intra-retinal. *Pre-retinal or subhyaloid hæmorrhages* are extravasations of blood between the retina and the vitreous. They always occur in the neighbourhood of the macula, and are usually large. They are round at first, but quickly become hemispherical, the upper margin being straight; this is due to the effect of gravity (Plate XIII., Fig. 1). Occasionally two such hæmorrhages may be seen in the same eye. Retinal vessels are hidden from view in the affected area. The upper layers become lighter in colour, generally attributed to the sinking of the red corpuscles. The blood gradually becomes absorbed, usually in a patchy manner, but finally disappears, though numerous cholesterin crystals may often be left as bright glistening spots. Vision is restored, but recurrences are

not uncommon, and other complications of vascular origin may modify the otherwise favourable prognosis. They are, for example, not uncommon in cases of subarachnoid hæmorrhage (*vide* p. 609).

Intra-retinal hæmorrhages, as already mentioned, are striate

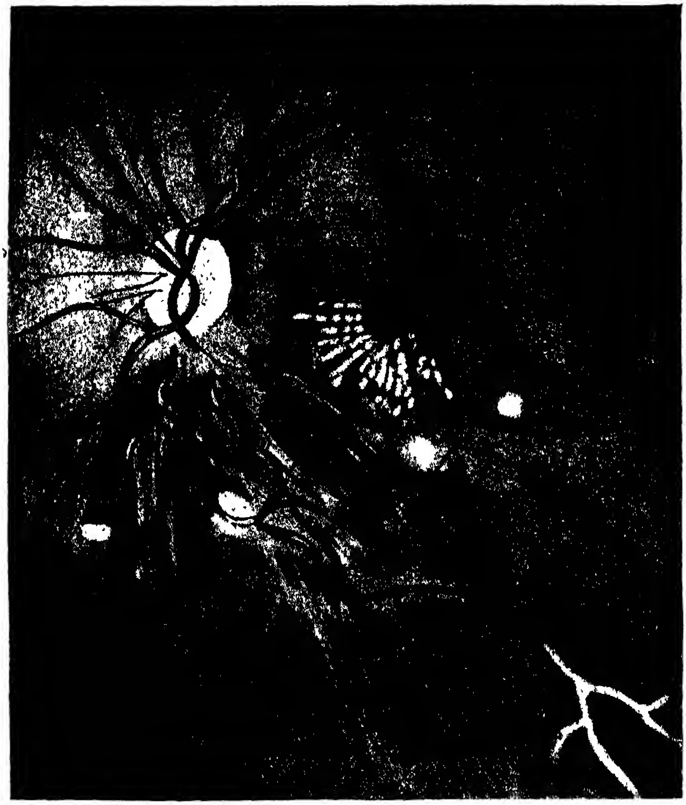


Fig. 200.—Thrombosis of a branch of the central retinal vein.

or flame-shaped when situated in the nerve fibre layer, rounded or irregular when in the deeper layers or between the retina and choroid. Intra-retinal hæmorrhages are absorbed very slowly, gradually becoming white, rarely pigmented.

Retinal hæmorrhages are due to many causes. Most frequently the vessel walls are weakened by general disease, which may be a vascular degeneration due to age or to altered

composition of the blood, as in pernicious anæmia, leucæmia, scurvy, purpura, nephritis, diabetes, hyperemesis gravidarum (*vide* p. 616), &c. Any of the causes leading to retinal hyperæmia may give rise, secondarily, to hæmorrhages. They may be due to pressure during birth in new-born infants, and are

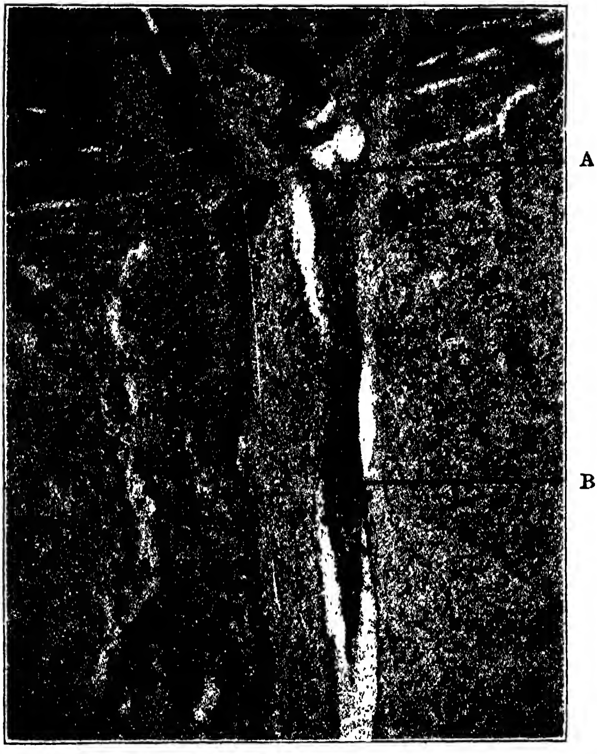


FIG. 201.—Thrombosis of central vein. (Coats.) Longitudinal section of nerve. From the region of the lamina cribrosa, A, backwards, the vein is occupied by a homogeneous coagulum; there is slight endothelial proliferation. At B the coagulum is invaded by cells, polymorphonuclear leucocytes, and larger paler cells.

probably responsible for some so-called congenital retinal defects, *e.g.*, white and pigmented spots of atrophy at the macula and elsewhere. Similarly they occur in cases of severe compression of the thorax or neck in older people. Whooping cough may lead to retinal as well as the more common conjunctival hæmorrhages. Traumatism, *e.g.*, severe contusions

and wounds, gunshot wounds of the eye, and of the orbit without direct injury to the eye, is another cause, and is often responsible for very extensive extravasations of blood, which may burst through into the vitreous. To this category belong the post-operative hæmorrhages. When the globe is opened in performing cataract extraction, or, still more, iridectomy for glaucoma, the normal or raised intraocular tension is suddenly reduced to zero. The intraocular vessels then dilate, and great strain is thrown upon their walls. If these are diseased, there is much danger of their rupturing. Considering the age and condition of these patients, it is surprising that hæmorrhage is not more frequent.

Minute hæmorrhages, unless in the macular region, may cause little obscuration of vision. Subhyaloid hæmorrhage usually abolishes central vision temporarily: it takes some weeks to clear up, the length of time varying with the size of the extravasation. The smaller spots may remain unaltered for months, though this is only apparent in some cases, old spots clearing up and being replaced by new ones. They may be absorbed without leaving any trace.

Venous Thrombosis may affect the central vein of the retina (Plate XII., Fig. 2) or one of its branches (Fig. 201). In the former case the obstruction is always just behind the lamina cribrosa (Fig. 201). All the veins of the retina become enormously engorged with blood, and extremely tortuous. Blood escapes from the capillaries at innumerable spots, so that the retina is covered with hæmorrhages. Sight is much impaired, though not so rapidly as in obstruction of the central artery, but recurrent extravasations finally destroy it entirely. In the early stages there is constriction of the field of vision and usually a central scotoma. When a single branch is blocked the œdema and hæmorrhages are limited to the area supplied by the vein; the block is usually at a bifurcation or where a sclerosed artery crosses the vein. In these cases the defect in vision is not sectorial, as in the case of a branch of the artery; the prognosis for central vision is best in these cases, but unfortunately it is the superior temporal vein which is most often blocked. The affected retina becomes atrophic, with fine pigmentary changes. Secondary glaucoma ensues in two to three months in a considerable number of the cases, probably owing to the increased albuminous constituents of the intraocular lymph. It does not occur when only a tributary is involved. In many cases bunches of tortuous new vessels are formed upon the disc

(Fig. 202); in others a collateral circulation is effected by similar tortuous new vessels in the retina. Such vessels often project forwards into the vitreous, and may rupture, leading to extensive vitreous hæmorrhages. Vascular disease and hæmorrhages are not infrequently present in the other eye, and bilateral thrombosis of the central vein sometimes occurs. In all cases examined microscopically the vessel walls show endothelial proliferation. The lumen is constricted, and it is probable that this factor is as important in causing obstruction as actual thrombosis.

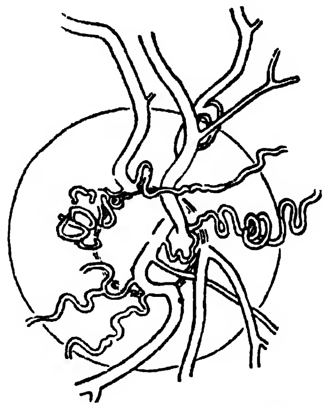


FIG. 202.—New-formed vessels. (Foster Moore.)

The patients are usually elderly, with cardiac or vascular disease, often induced by nephritis; the blood pressure is usually high, and there is often albuminuria. Women are affected more often than men. The obscuration of vision is almost always noticed on waking in the morning. Probably the low blood pressure and sluggish flow during sleep allow thrombosis to occur in the vessel where it is constricted normally at the lamina cribrosa and pathologically by endovascular changes. Thrombosis may, however, be due to local causes, such as orbital cellulitis, following facial erysipelas, &c. It occasionally occurs in young people following a febrile attack, and is then probably due to an infective phlebitis. The other eye usually shows vascular disease and often small hæmorrhages in the periphery of the retina. Bilateral thrombosis is rare.

Treatment. No treatment is of avail in cases of complete occlusion. Cases of improvement in partial thrombosis by intravenous injections of heparin have been published. Atropine should be avoided, as tending to induce glaucoma. Local treatment is indicated only if the eye becomes painful, when it should be removed. The condition is to be regarded as a danger signal, and constitutional investigation and treatment should be carried out assiduously.

"Retinitis Proliferans." When hæmorrhage occurs into the vitreous the blood clot is usually almost completely absorbed. This is probably due to the absence of fibroblasts in the

vitreous and their scantiness in the retina, for the retinal connective tissue consists of neuroglia, an epiblastic structure which probably takes no part in fibrous tissue formation. The only mesoblastic tissue in the retina is that forming and surrounding the retinal blood vessels. In some cases, however, blood clot may organise, giving rise to masses of fibrous tissue in the vitreous, vascularised by newly formed blood vessels derived from the retinal system (Plate XIII., Fig. 2). This condition is known as "retinitis proliferans." The tissue is most commonly situated near the disc, and the vessels spring from this neighbourhood, probably owing to the fact that there is more mesoblastic tissue here than in other parts of the fundus.

There seems to be some special factor necessary to stimulate organisation; and it is found in some general diathesis. In nearly all these cases there is either a history of syphilis or the patient is suffering from nephritis, diabetes, or some other form of toxæmia, the origin of which may be obscure, as in some cases of recurrent hæmorrhage (*vide* p. 332).

The amount of fibrous tissue varies from the most delicate strands and films supporting new-formed blood vessels—specially common in syphilitic cases (*vide* p. 372)—to dense bands and membranes stretching far forwards into the vitreous and hiding the fundus. Most extensive proliferations are met with in war injuries with rupture of both choroid and retina. The bands are often attached to the retina at their apices, and as the fibrous tissue contracts after formation the retina may be pulled up and detached.

Vision is generally greatly impaired and often lost, usually from detachment of the retina. No treatment is of any avail, but attention should be given, where that is possible, to the constitutional cause of the hæmorrhage.

Retinitis Circinata. (*Syn.—Circinate Degeneration*). Retinal hæmorrhage may give rise to yet another condition in rare cases, generally elderly women. In retinitis circinata there is a girdle of bright white patches with crenated borders around the macula (Fig. 203). The diameter of the girdle, which is usually an imperfect circle or ellipse, or horseshoe-shaped, open towards the temporal side, is generally considerably greater than a papilla diameter, and follows the larger macular branches of the superior and inferior temporal vessels. The vessels pass over the spots. The macula shows yellowish white areas, slight pigmentation and often hæmorrhages. The patches develop slowly and are

usually well advanced before noticed. The disease is unilateral in about half the cases; exudative retinitis has been observed in the other eye (Coats). Central vision is much reduced, but the field remains full The patches sometimes disappear slowly

FIG. 203.—Retinitis circinata. (Holmes Spicer.)

and vision improves. Mercury and iodides seem to afford the best chance of amelioration.

Exudative Retinitis (*Syn.—Massive Exudation in the Retina* (Coats)) resembles conglomerate tubercle in its ophthalmoscopic signs. There is usually a large raised yellowish white area or several smaller areas posterior to the vessels. The vessels often show gross degenerative changes, and there is sometimes arterio-venous communication, with enormous dilatation of the veins. The patients are usually boys, otherwise apparently healthy. Detachment of the retina, cataract or glaucoma may occur in the late stages. There is always evidence microscopically of hæmorrhage between the retina and choroid and in the deep layers of the retina; the choroid is at first healthy.

Angiomatosis of the Retina (*Syn.—Angiomatosis Retinæ*, von Hippel, Lindau) is a rare familial disease which generally becomes manifest in the third and fourth decades of life, more frequently in males than females. In its later stages it resembles exudative retinitis in ophthalmoscopic appearance. The cerebellum, medulla, spinal cord, kidneys and adrenals are also affected with angiomatosis and cysts. The ocular lesions are often bilateral, slowly progressive, and may precede a fatal cerebellar lesion by ten to fifteen years. The ophthalmoscopic appearances vary:

(1) A raised pinkish-yellow swelling about 3 mm. in diameter, situated between the equator and the ora serrata, to and from which run a branch of the central artery and vein, both dilated to three or four times their normal calibre and very tortuous. (2) Multiple crimson glomerulus-like tufts at the ends of tortuous arterioles, situated between the optic disc and the equator. (3) A mulberry-like swelling containing small cysts on the disc and adjacent retina. Retinal and vitreous hæmorrhages and

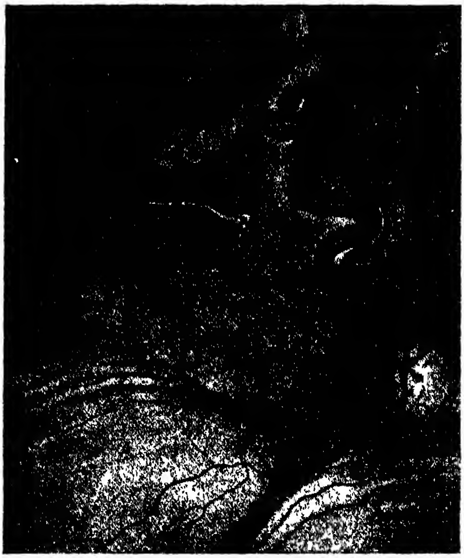


FIG. 204.—Angiomatosis retinæ. (Stallard.)

retinal exudates occur later, and detachment of the retina leads to blindness.

Treatment. In the early stages the insertion of a katholysis or diathermy needle into the retinal lesion is effective in destroying it. The number of perforations is assessed according to the size of the mass.

Renal Neuro-retinitis (*Syns.—Renal Retinopathy, Albuminuric Retinitis*) in its most typical form, presents an ophthalmoscopic picture which is almost pathognomonic, being simulated only in some cases of intracranial tumour (Plate XIV.). In addition to the general signs of retinitis—haziness of the retina and disc, hyperæmia and hæmorrhages—the distinguishing feature is the presence of brilliant white spots

and patches in the retina. The earlier deposits are cloudy, with soft edges ("cotton-wool" patches); the later brighter, more sharply defined and punctate. The disc is surrounded by large white patches or by a continuous "snow-bank." Around the macula are smaller dots or round patches, also silvery white. Radiating from the fovea are spokes of white dots or fine lines, forming a star-shaped figure which is extremely characteristic. The fovea itself escapes and the star is often incomplete in some direction. The vessels generally show very definite degenerative changes (*vide* p. 357). In some cases, especially in the albuminuric retinitis of pregnancy, a flat detachment of the retina occurs, almost certainly due to the retina being raised from the choroid by exudates. The detachment is usually bilateral and involves the lower part of the fundus. Unlike most detachments of the retina, these frequently disappear, the exudates being absorbed.

FIG. 205.—Renal retinitis. Masses of fibrinous exudate in the retina.

Renal retinitis by no means always displays the typical picture. Often there is a neuro-retinitis which exhibits no characteristic features specially associated with nephritis. In these cases there is moderate swelling of the disc—seldom so much as in the choked disc of intracranial disease—more or less widespread œdema and hæmorrhages. Irregularly scattered bright white spots and patches may be present or wanting. *The urine should be examined in every case of retinitis.*

The white spots of renal retinitis are chiefly composed of exudates, which are often fibrinous (Fig. 205), later becoming hyaline. They are situated particularly in the outer reticular layer, where large vacuoles are filled with fluid, fibrinous coagula, or hyaline deposits, often associated with large globular macrophages; they may be present in all the layers. There is some leucocytic infiltration, and peculiar swollen nucleated structures—cytoid bodies—are found in the nerve fibre layer; they are probably varicose nerve fibres. The exudates and necrotic retinal elements undergo fatty degeneration. The peculiar arrangement of the spots in the macula

is not due to any supposed radial distribution of Müller's fibres in this situation, but to fluid which raises the internal limiting membrane and follows the radial course of the nerve-fibres as they arch towards the disc. Spread of the œdema to the loose reticular layer, which is very well marked in this situation, throws the retina into actual radial folds, the fovea itself remaining unchanged and as it were "pegged down." The disc shows the same changes as in papillœdema (*q.v.*), but less marked. The changes in the blood vessels are those common to vasculitis in other small vessels, consisting of endo- and peri-vasculitis and hyaline degeneration of the walls.

There is no constant relationship between the retinitis and the vascular disease. The vessels may be quite normal, *e.g.*, in some puerperal cases and in the rare cases occurring with acute nephritis. In the cases with chronic interstitial nephritis, *i.e.*, the great majority, the vessels are much diseased. The retinitis is, therefore, not directly due to the vascular disease, but probably to toxins circulating in the blood stream.

Renal retinitis occurs in about one-third of cases of nephritis and may occur in all forms of nephritis, including scarlatinal, puerperal and "trench" nephritis, but in by far the greatest number of cases the disease is chronic interstitial nephritis, and it is very rare in ordinary acute nephritis. The last-mentioned fact accounts for the small quantity or even total absence of albumin in the urine in some cases. Though the degree of retinitis bears no fixed relationship to the nature or severity of the renal mischief, yet in all cases its presence is of grave significance. As a general rule its appearance requires a combination of two essential factors—toxæmia and hypertension with vascular sclerosis; it is seen in its most marked form in malignant hypertension (nephro-sclerosis). The retinal changes may be the first evidence of renal disease, hence the extreme importance of their discovery, which is accentuated by the fact that the majority of hospital patients die in from six months to two years. Under more favourable conditions patients may survive five years or even longer. The prognosis is equally grave in children. The risk to life is much less in the scarlatinal and puerperal cases. In the later the prognosis is worse the earlier the onset of the retinitis, but fortunately it seldom commences before the sixth month of pregnancy. The artificial induction of abortion is indicated, and usually has a prompt beneficial effect; vision, however, is usually permanently impaired, the

PLATE XIV.

FIG. 1.—Renal neuro-retinitis

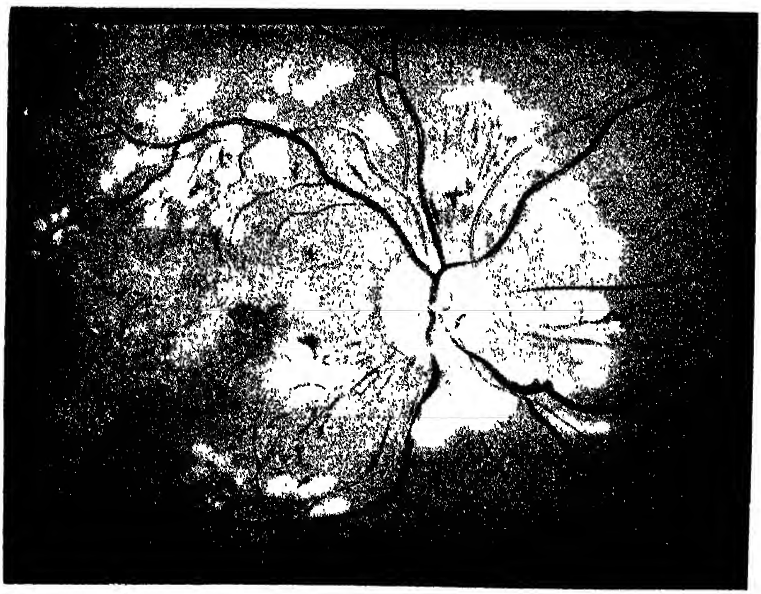
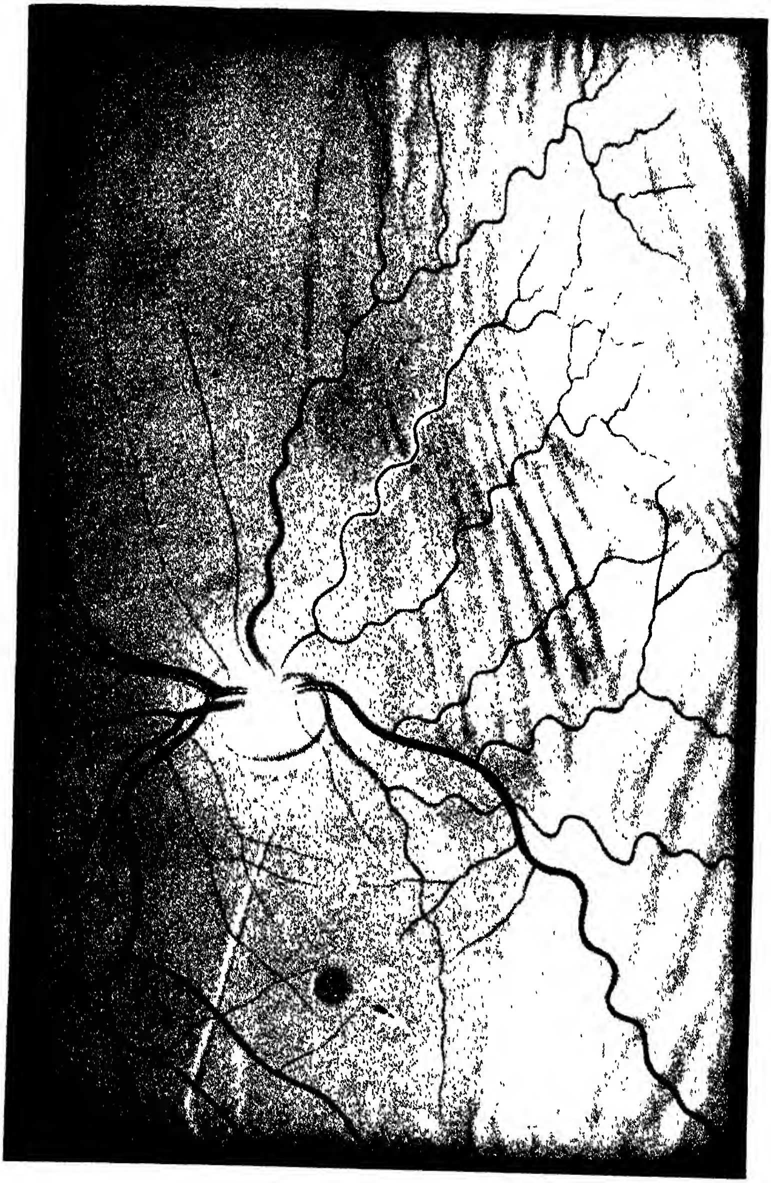


FIG. 2.—Renal neuro-retinitis.

[To face p. 368.

PLATE XV.



Simple detachment of the retina (diagrammatic).

degree depending upon the duration of the retinitis. The "cotton-wool" patches clear up first, the glistening macular spots more slowly. Partial optic atrophy and slight retinal changes, such as white or pigmented spots at the macula, follow. The disease does not always occur at the first pregnancy; but may, after one attack, recur at subsequent pregnancies, though by no means always. The patient should, however, be warned of the danger. The retinal changes in puerperal cases are usually severe, and detachment of the retina occurs more frequently in these than in other cases. If pregnancy is not interrupted spontaneously or artificially death (about 15 per cent. of cases) or blindness (about 13 per cent.) occurs, and the child is usually born dead.

Diminution of visual acuity is commonly the only symptom complained of, and, as mentioned, may lead to the discovery of the renal disease. Generally the history of severe headaches can be elicited, and the blood pressure is high, usually about 200 mm. Hg. The condition is almost always bilateral. It very rarely causes complete blindness. It occurs more often in men than in women, owing to the greater incidence of nephritis in men; in children it is commoner in girls than in boys. It is commonest between thirty and sixty years of age, especially in the last decade; but it is probably more frequently associated with the parenchymatous nephritis of children, which is generally syphilitic, than has been hitherto thought (Nettleship).

It is important to note that transient blindness may occur in the course of nephritis, especially associated with uræmia. In these cases the retina shows no abnormality, or at most changes which may occur independently of the disease. This *uræmic amaurosis* is distinguished in being sudden total blindness, whereas the defective vision of albuminuric neuro-retinitis is slower in onset and never complete. Sight usually returns in one or two days (*vide* p. 414).

Diagnosis. The typical picture of albuminuric retinitis may be nearly simulated in some cases of increased intracranial pressure, particularly when due to cerebral tumours occurring in children (*vide* p. 388). It may occur without any definite signs of nephritis, and somewhat similar appearances are met with in diabetes and leucæmia. Rarely a star at the macula, with or without slight papillitis, has been met with in young persons with anæmia or chlorosis, or without discoverable cause.

The *treatment* of these conditions is purely constitutional.

Diabetic Retinitis. Retinitis is a relatively rare complication of diabetes, occurs in the late stages, and in elderly people; it is not improbable that it is frequently missed owing to the peripheral position of the lesions, opacities of the lens, &c. It is generally, but not always, bilateral. Irregularly scattered small, bright white spots around the macular region are the commonest manifestation. The snowy patches and stellate

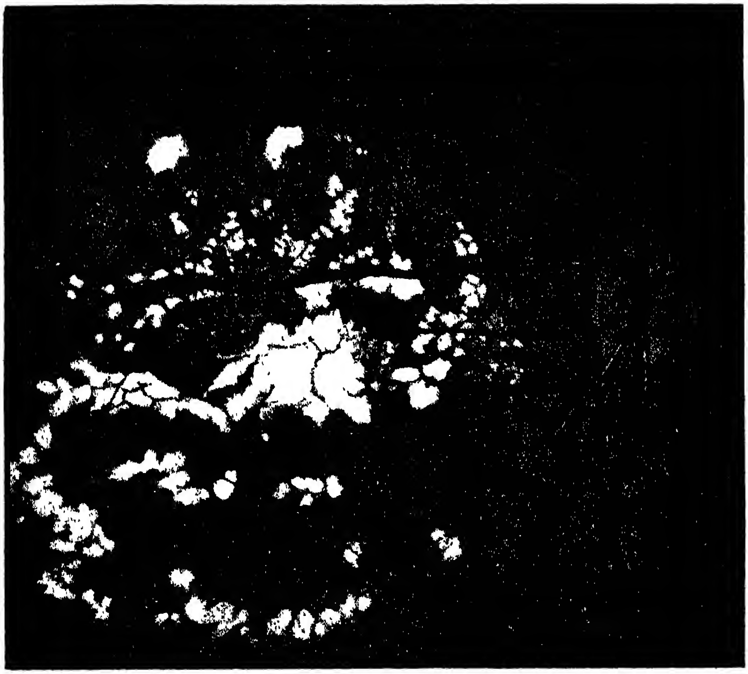


Fig. 206.—Diabetic retinitis.

arrangement at the macula are usually absent, but it must be remembered that albuminuria is a frequent concomitant of the late stages of diabetes, and all the characteristics of albuminuric retinitis may occur. The white spots may coalesce into larger plaques with crenated edges, which indicate their mode of formation. Punctate hæmorrhages are freely scattered over the fundus: they are more often round and deeply seated than linear and superficial, as in renal retinitis. The vessels are often normal, but a degree of vascular degenera-

tion correlated with the age of the patient is not uncommon: it is noticeably less than in albuminuric retinitis. The optic disc is generally normal, as well as the remainder of the retina.

The prognosis depends upon the severity of the constitutional condition; 60 per cent. live more than two years. Under insulin treatment the retinitis may persist indefinitely, for this treatment appears to have no effect upon it.

Diabetic retinitis cannot be due to sugar, di-acetic acid or acetone circulating in the blood, since it does not occur in children in whom these products of perverted metabolism are most markedly present. The minimal pathological change is the deposition of fatty droplets in the endothelial lining of the retinal capillaries near the macula. Where these droplets are most numerous they tend to obstruct the lumen of the vessel, forming a small micro-aneurysm. This micro-aneurysm in the macular area is the earliest retinal diabetic change seen with the ophthalmoscope, and hitherto it has been mistaken for a retinal hæmorrhage. The venous obstruction thus caused in turn gives rise to changes throughout the retinal venous system, varying from networks of newly formed vessels to gross beading and looping of the larger veins (Ballantyne).

A peculiar feature sometimes met with in diabetes is *lipæmia.* It occurs especially in young patients, with marked acidosis, and the prognosis is grave. The ophthalmoscopic appearances are then striking, the retinal vessels containing fluid which looks like milk. The arteries are pale reddish, the veins having a slight violet tint. The general fundus has much the normal coloration. Lipæmia responds rapidly to insulin treatment.

Leucæmic Retinitis. When retinal changes occur in the course of leucæmia the ophthalmoscopic appearances are characteristic. The fundus is pale and orange-coloured. The veins are dilated and tortuous, often with white lines along them, and are bright red, not dark; the arteries are small and pale yellowish red. Very typical are white spots and patches surrounded by a red rim; they consist of leucocytes surrounded by red corpuscles. These are present only rarely, and are also found in pernicious anæmia. They are most common in the periphery of the retina. In every doubtful case the blood should be examined.

Special Forms of Retinopathy

Syphilitic Retinitis. Syphilis is one of the commonest causes of retinitis, but it is usually a secondary retinitis, accompanying

disease of the choroid. It, however, occurs as a primary retinitis also, and in this form syphilitic endarteritis is a prominent sign. There are dust-like opacities in the vitreous, especially in the posterior part; the retina is cloudy, particularly in the neighbourhood of the disc, which may be hyperæmic. White spots may be seen in the macular region, and yellowish or white spots, often bounded by pigment, at the periphery of the fundus. The vessels may be degenerated (*vide* p. 357), with whitish exudations along their course; hæmorrhages are rare.

Every transition is seen to a condition much resembling retinitis pigmentosa (*vide* p. 375), but seldom showing such a characteristic distribution of the pigment.

The subjective symptoms are defective central vision, night-blindness, irregular and concentric contraction of the field, with or without central, paracentral, or ring scotomata, and metamorphopsia.

In most cases the amount of organisation which takes place at the sites of the inflammatory deposits of syphilitic retinitis is very small, but in some cases there is a well-marked tendency to the formation of new blood vessels. These may not be limited to the retina itself, but may extend into the vitreous, forming convoluted coils. They are held together by a minimum of delicate connective tissue (*cf.* Retinitis proliferans). They are commonest on or near the disc.

In acquired syphilis the disease usually occurs one to two years after infection; usually both eyes are involved, but not with simultaneous onset. As a rare manifestation the macular area is alone affected, showing a grey or yellow deposit, or numerous small yellow spots and dots of pigment. This form shows a great tendency to relapse.

Retinitis is not uncommon in congenital syphilis. Such patients often show a dusty or peppery discrete pigmentation of the retina at the periphery, associated with a tigroid condition of the fundus in this situation. It is only distinguishable from what is often seen as a normal condition by the greater aggregation of the pigment. There may be thickly strewn black and white spots, like a mixture of pepper and salt. In more definite forms there are yellowish red and black spots at the periphery (anterior retinitis), a condition often seen in interstitial keratitis (*vide* p. 238), or larger grey or white patches may be seen, or the condition observed in the acquired form may be fully developed.

If the diagnosis is doubtful the Wassermann test should be applied.

Treatment. A prolonged course of anti-syphilitic treatment is indicated. Dark glasses should be worn and the eyes rested.

Septic Retinitis (Roth). Apart from endogenous retinitis (*vide* p. 343), metastatic retinitis may manifest itself in the form of small round or oval white spots near the disc, often accompanied by hæmorrhages. This form occurs in puerperal and other forms of septicæmia. Usually both eyes are affected.

Purulent Retinitis. This is most commonly due to a septic perforating wound, being then a precursor of panophthalmitis (*vide* p. 462). Rarely it is a metastatic condition, occurring in pyæmia, and probably starting in a septic embolus. In the early stages there is severe retinitis with hæmorrhages. Suppuration rapidly follows, involving the vitreous, so that a yellow reflex is obtained. The condition often passes into panophthalmitis, but less commonly than in cases of exogenous infection. Pyogenic organisms are attenuated in the blood stream and tissues, so that the process may subside with the restoration of useful vision (*vide* p. 343).

Toxoplasmosis. Toxoplasmosis, a protozoan infection, was first encountered in infants as a severe disseminated encephalomyelitis (Wolf, Cowan and Paige, 1941). Rodents and birds are the most likely source of the infection. Lesions occur in the eyes with considerable frequency, the most typical of which are bilateral and frequently multiple areas of chorio-retinitis of an inflammatory and necrotising type similar to those found in the brain. The most characteristic lesion occurs at the macula where a deep punched out scar is left resembling the clinical appearance of a congenital macular coloboma. A uveitis may also occur.

Retinitis from Bright Light, which might be termed *photoretinitis*, occurs after exposure of the unprotected eyes to bright sunlight, as in looking at an eclipse of the sun with unprotected eyes (" eclipse blindness "), or the electric light, as in the intense flash of the short-circuiting of a strong current. The relative parts played by the different rays of the spectrum are not fully understood. Only a few, relatively innocuous, ultra-violet rays reach the retina. On the other hand, practically all the visible rays and many infra-red rays pass unimpeded to the retina (*vide* p. 23). Much of this radiation is absorbed by the pigment epithelium, and it is probable that the pathological changes are produced by the resultant heating effect. It is, in fact, a burn of the retina.

The symptoms are persistence of the after-image, passing on later into a positive scotoma, and metamorphopsia. Ophthalmoscopically there may be no signs at first, or a pale spot is seen at the fovea with a brownish red ring round it. Later there are usually deposits of pigment and small grey punctate spots around the fovea. Prognosis must be guarded, since, though improvement often occurs, some defect usually remains, and the scotoma may persist permanently.

The *treatment* is that of retinitis in general. Smoked glasses

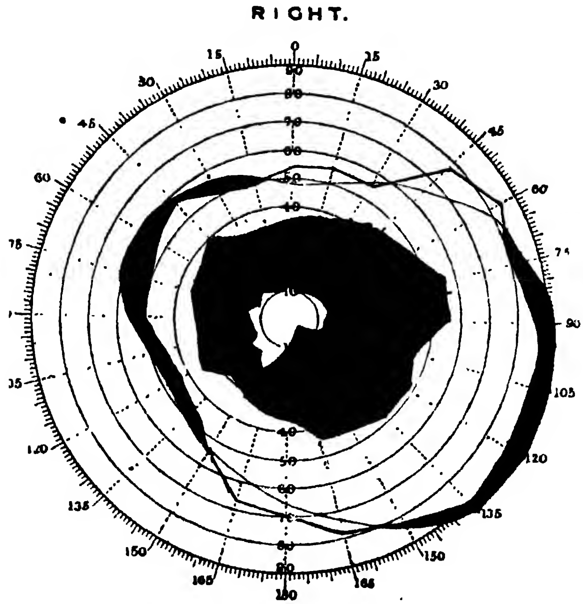


FIG. 207.—Ring scotoma, from a case of retinitis pigmentosa.

should be used, or Crookes's glasses, *i.e.*, such as cut off the ultra-violet rays.

Retinitis pigmentosa (*Syn. Pigmentary Degeneration of the Retina*) is a slow degenerative disease of the retina in both eyes, beginning in childhood and often resulting in blindness in middle or advanced life. The degeneration commences in a zone near the equator of the eye, and gradually spreads both anteriorly and posteriorly. The macular region is not affected until very late in the disease. The condition is bilateral. The peculiar site of origin has been attributed to the relatively

weak choroidal circulation in this zone, which is the meeting place of the short ciliary and the recurrent ciliary arteries (*vide* p. 13)

The symptoms of the disease are very characteristic, the most prominent being defective vision in the dusk (night blindness) (*vide* p. 417). This symptom may be present several years before pigment is visible in the retina. Vision under low illumination is carried on essentially by the rods (*vide* p. 68), and night-blindness is a sign of defective nutrition of these structures.

Examination of the vision may show perfect central visual acuity. Investigation of the field of vision, seldom satisfactory in children, but more reliable in young adults, shows concentric contraction of the fields, specially marked if the illumination is reduced. In early cases a partial or complete annular or ring scotoma is found (Fig. 207), corresponding to the degenerated zone of retina. As the case progresses the field becomes continually slowly smaller, until at last it is reduced to a small area round the fixation point. Central vision may even now be normal, but the patients are quite incapacitated from getting about, for they are in much the same condition as a person looking down two long cylinders—they see only the thing they are actually looking at and nothing around. They therefore grope about helplessly. Loss of central vision does not usually occur until fifty or sixty years of age, but vision may be lost earlier through cataractous changes occurring in the centre of the posterior cortex of the lens.

Ophthalmoscopic examination shows also a very characteristic picture (Plate XI.). In the zone affected the retina is studded with small jet-black spots, resembling bone corpuscles or spiders. The retinal veins, never the arteries, often have a sheath of pigment for part of their course (*vide* p. 346; Plate XI., Fig. 2). As the pigment from the retinal pigment epithelium migrates into the superficial layers the epithelium itself becomes decolorised, so that the choroidal vessels are now visible and the fundus appears tesselated. The pigment spots which lie near the retinal vessels are seen to be anterior to them, so that they hide the course of the vessels. There is no question, therefore, as to their exact position in the retina itself. In this respect they differ from the pigment around spots of choroidal atrophy (*vide* p. 338), in which the retinal vessels can be traced over the spots. The number of pigment spots differs much in individual cases, and they are often very

scanty in the early stages, at which time attenuation of the retinal vessels is frequently the most striking feature.

In the earliest stages it can be proved that it is a zone of the retina which is affected and not the most anterior part, for normal retina may be seen at the peripheral limits of the ophthalmoscopic field. In the later stages this area diminishes *pari passu* with extension of the disease towards the centre.

The retinal blood vessels, both arteries and veins, become extremely attenuated and thread-like.

As the pigmentation increases and the retina becomes more and more atrophic the ganglion cells are destroyed, thus leading to degeneration of their axis cylinders, viz., the fibres in the nerve fibre layer of the retina and the fibres in the optic nerve. Optic atrophy, therefore, sets in and gradually increases. The disc exhibits the characteristics of primary optic atrophy (*vide* p. 401), but is not quite typical of this condition. The disc is pale, but seldom more than yellowish white, like wax: there is no excess of fibrous tissue, but the vessels are very small and thread-like.

In the later stages the nutrition of the lens suffers. The complicated cataract which is formed is a very typical form of progressive posterior cortical cataract, going on to complete opacification of the cortex.

The cause of retinitis pigmentosa is unknown. Several members of the same family are often affected, consanguinity of the parents is not infrequent, and the disease may affect several generations. Nettleship found heredity without consanguinity in 23 per cent., consanguinity without heredity in 23 per cent., and heredity combined with consanguinity in 3 to 4 per cent. Males are more frequently affected than females. The patients or other members of the family are found not infrequently to suffer from insanity, epilepsy, or other signs of mental debility. About one-third of the patients are deaf, and 4 per cent. of deaf mutes have retinitis pigmentosa (Nettleship). Evidences of cerebral disease or of congenital anomalies in the eyes or other parts of the body (harelip, &c.) may be present: it may also be associated with obesity, hypogenitalism, tetany, mental defect and polydactyly (Laurence-Moon-Biedl Syndrome). Congenital syphilis may produce similar results, though the distribution of the pigment spots is seldom quite typical (*vide* p. 346).

In syphilitic choroido-retinitis exactly the same ophthalmoscopic appearances associated with night-blindness may occur and may be present in both eyes. These cases are rare

and can be distinguished by the later onset of night-blindness and the absence of the disease in other members of the family. Much more commonly in syphilis the patches are limited to certain areas of one or both retinæ. The prognosis is better in the syphilitic cases, since they may become stationary. It is best to restrict the term "retinitis pigmentosa" to the very typical congenital and familial disease.

Pathology. Few cases of retinitis pigmentosa have been examined microscopically, and those usually at a very late stage. Section of the posterior ciliary vessels leads to pigmentation of the retina in rabbits (Wagenmann), but the retinal changes differ materially from those in retinitis pigmentosa (Nicholls). Doubt has therefore been cast upon the choroidal origin of the disease; and in some advanced cases the choriocapillaris has been found normal. Retinitis pigmentosa has been found as an hereditary Mendelian recessive in rats (Bourne, Campbell and Tansley). In it the rods degenerate first, showing the earliest signs in the nuclei; and vascular sclerosis of both retina and choroid only occurred later. The evidence, therefore, seems to be that the disease is a primary retinal degeneration, though its frequent association with deafness and cerebral disorders suggests that it is a local manifestation of a congenital weakness of tissues of the central nervous system of unknown origin—a so-called abiotrophy.

Treatment is eminently unsatisfactory, since nothing appears to have a decided influence upon the course of the disease. Trephining at the corneo-scleral margin has been followed by temporary improvement (Mayou), probably by improving the intraocular circulation. Extraction of the lens may be followed by some improvement in vision, but should not be done until a late stage is reached. Attention should be paid to the general nutrition, since it has been proved that defective formation of visual purple, and consequent night-blindness, may follow a diet poor in vitamin A; hence cod-liver oil, halibut oil, or carotene in oil should be given; also Ca lactate and vitamin D. Attempts to improve the circulation by vasodilators, *e.g.*, acetylcholine, and by removal of the superior cervical ganglion have proved useless.

Retinitis pigmentosa sine pigmento is a variety of the disease with the same symptoms, but without visible pigmentation of the retina. It is almost certainly only the early stage of the ordinary disease (Nettleship). It is progressive and leads to optic atrophy, therein differing from *congenital night-*

blindness, which is a rare hereditary disease without ophthalmoscopic signs, remaining stationary throughout life.

Allied to these conditions is *retinitis punctata albescens*, in which, with the same history and symptoms, the retina shows hundreds of small white dots distributed fairly uniformly over the whole fundus. The condition is almost certainly an atypical variety of retinitis pigmentosa, and may occur in a family affected with this disease. It differs in the important fact that it is almost if not quite non-progressive, but one case is on record in which the white spots disappeared and typical pigment spots developed (Nettleship).

Detachment of the Retina (*Syns.—Ablatio retinæ, Amotio retinæ*). The retina may become separated from the choroid by being pulled up from within or pushed up from without. The simplest example of the former mode is in the late stages of plastic cyclitis when the strands of connective tissue which become attached to the retina contract during the process of organisation. The simplest example of the second mode is separation by means of a choroidal hæmorrhage, such as may occur from a blow, or disease of the vessels.

Clinically detachment of the retina is observed most commonly in three conditions, viz., after a blow, in high myopia (60 per cent. of cases of detachment), and in sarcoma of the choroid; to these must be added a not inconsiderable number of cases in which no cause can be assigned. The exact mechanism of detachment in these cases is by no means completely understood; indeed, detachment of the retina is still one of the difficult problems of pathology. It is commonest in men between forty-five and sixty-five years of age.

The patient usually complains that there is a cloud in front of one eye, so that parts of objects, usually the upper or lower parts, are not seen. In other words, there is a positive scotoma, as is confirmed by making a chart of the field of vision. Usually the scotoma corresponding to the detached area is absolute, but in shallow detachments some vision may persist, sufficient nourishment being afforded to the retina from the subretinal fluid. There is then generally a relative scotoma for colours, and acquired colour-blindness of the tritanopic type (*vide* p. 419) is not uncommon. It is well to take the field under high and low illumination, the increase in the size of the scotoma in the latter case showing that a larger area of the retina is involved than that which is completely blind. As a rule central vision is intact at first, but all detachments of the retina tend in time to be complete; when the

macular region becomes affected central vision is lost, and when the detachment is total perception of light is lost. The first symptom observed sometimes is transient flashes of light (photopsiæ), due to slight displacements of the retina which irritate the neuro-epithelium. They should always be regarded with serious attention, but not infrequently occur, especially in myopic eyes, without being followed by detachment.

A small detachment causes much less definite signs. Some obscuration of vision is noticed, but the diagnosis can only be arrived at by careful examination of the fundus and of the field of vision. In sarcoma of the choroid the detachment may be very small and in any position, thus differing from the other forms—so-called *simple detachment*—in which it is generally larger, though often shallow, and confined to the lower parts of the fundus. Simple detachment often begins in the upper part of the fundus, but after a variable time the subretinal fluid gravitates to the lower part of the eye and the retina becomes replaced in the upper part. Sarcoma of the choroid may start in the neighbourhood of the macula, and in this case central vision is early affected, as shown by distortion of objects (metamorphopsia, micropsia, &c.) or a relative scotoma for colours. A small detachment due to a sarcoma of the choroid may be accompanied by a large simple detachment in the usual situation below.

Externally the eye looks normal; the anterior chamber may be deep and the tension diminished, though rarely much in the early stages. In cases due to sarcoma of the choroid the tension is always raised in the later stages and the anterior chamber is shallow; occasionally the tension is raised early in these cases.

It is by no means difficult to miss diagnosing a detachment of the retina even when it is large, especially if it is also shallow. The symptoms may be indefinite, for the retina may obtain sufficient nourishment from the fluid which underlies it to retain its functions only partially impaired for a considerable period. Failure in diagnosis is almost always due to the omission of a proper routine examination of the eye. The observer often employs the direct method, possibly after a casual examination by the indirect, without previously examining with the mirror alone. A shallow detachment will then appear little altered from the normal fundus. It is true that it is more hypermetropic, but the observer does not realise that he is exercising rather more accommodation in looking at it than at the rest of the fundus. Now, if the eye is examined with the mirror alone at ordinary reading distance, although

perhaps no details of the retina are seen—and they will only be seen if the retina bulges far away from the choroid—yet some difference in the nature of the reflex as the eye is turned in various directions will at once arrest attention.

Further examination by the direct method will then show the following changes (Plate XV.). The detached portion of retina has a different tint from the normal fundus. In the most typical condition it is quite white or grey, with folds which show a bright sheen at the summits and appear greenish grey in the depressions. During slight movements of the eye the folds show oscillations. The retinal vessels are seen coursing over the surface. They naturally follow all the curves of the folds. Very striking is their abnormal colour. Owing to the fact that they are separated from the choroid, which is responsible for the red reflex of the normal fundus, they are under much the same conditions as an ordinary vitreous opacity, *i.e.*, they cut off the light reflected from the choroid. They therefore look much darker than usual, and may be almost black. They show no central light streak and appear smaller than normal.

If now this portion of the fundus is observed with the highest convex glass with which it can be seen plainly, it will be found that the rest of the fundus is out of focus; this proves that it is displaced forwards and must therefore be detached from the choroid.

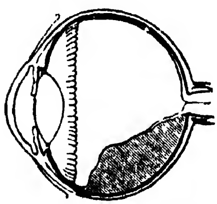


FIG. 208. — Diagrammatic sagittal section of eye with partial simple detachment of the retina. (Nettleship.)

In the early stages, and sometimes for a long period in shallow detachments, the colour differs little from the normal. Such cases are much less easy to diagnose, and may give rise to great difficulty, especially if the apparent detachment is very peripheral, for the periphery even of the emmetropic eye is usually seen best with a low convex glass.

When the detachment is very extensive great balloon-like folds may be seen, and these will probably cut off all view of the disc. At the edges of the detachment a considerable degree of pigmentary disturbance may be seen. White spots of exudation, hæmorrhages, and greyish-white lines due to retinal folds may be seen on the surface of the detached retina. Not infrequently a hole is visible, through which the bright red choroid can be seen (Figs. 209, 210). It is probable that there is a hole in every detached retina, but it is not always

visible ophthalmoscopically. The most frequent are horseshoe or arrow-head shaped, with a lid-like tongue. These are always peripheral and commonest in the upper parts of the retina. They are attributed to traction by vitreous bands.

FIG. 209.—Round hole.

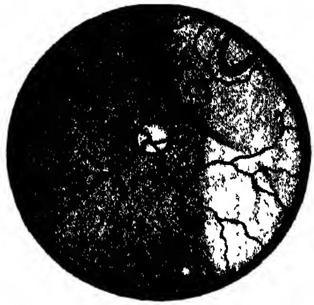


FIG. 210.—Arrow-head rent.

FIGS. 209 and 210.—Holes and the Retina. (Shapland.)

Round holes are usually less peripheral, and may occur at the macula. In some, especially traumatic, cases the retina becomes detached at the ora serrata (anterior retinal dialysis, "disinsertion of the retina") (Fig. 211), probably following cystic degeneration, which is quite common in this situation in old people. In these cases there is often a very large aperture through which the choroid can be seen, the edge of the detached retina being sharply defined. Disinsertion is commonest in the lower parts of the retina.

FIG. 211.—Anterior dialysis. (Shapland.)

In total detachment the retina is umbrella-shaped, remaining attached at the disc and at the ora serrata. Still later it becomes bunched up behind the lens, the part attached to the disc being pulled out into a straight cord. In these cases the disturbance to nutrition of the eye leads to the development of a complicated cataract (*q.v.*) so that ophthalmoscopic examination becomes impossible.

The description given applies especially to so-called *simple* detachment, *i.e.*, detachment not due to sarcoma of the choroid. The difference may be slight, but accurate diagnosis

is of the utmost importance, since the life of the patient may depend upon it. The chief diagnostic features are given in treating of sarcoma of the choroid, and should be very carefully studied (*vide* p. 425).

The space between the retina and choroid is filled with a highly albuminous fluid secreted by the choroid (Fig. 208).

The prognosis in simple detachment of the retina, untreated by operation, is unfavourable. The detachment becomes total, and complicated cataract and iridocyclitis follow. Spontaneous replacement is rare except in cases of renal retinopathy (*vide* p. 367). The results of surgical treatment are good in 80 to 90 per cent. of cases due to trauma with retinal dialysis at the ora serrata in the lower temporal quadrant. In healthy patients whose vitreous, retina and choroid show no disease other than changes at the site of the retinal hole the prognosis is good in about 75 per cent. of cases if operated on early. The prognosis is bad if the detachment has been present for nine months or more; when the vitreous, retina and choroid are degenerated; when there is high myopia; and always in restless and neurotic patients. Detachment recurs in some cases, even if the retina has remained *in situ* as long as one or two years.

Detachment in a myopic eye is an indication for extreme care of the other eye, which must be regarded as predisposed to the same accident. The patient should be warned against stooping, as in gardening, and lifting heavy weights.

Treatment. A thorough investigation of the affected eye is made before operation. Full dilatation of the pupil is very important: in order to reveal a retinal tear at the ora serrata it may be necessary to inject mydricain (3 minims) under the conjunctiva near the limbus in the lower part of the globe. Sometimes such a lesion is rendered visible only by making gentle pressure on the sclera near the ora serrata with a strabismus hook. A careful drawing, showing the position of retinal holes, pathological lesions, retinal vessels and other landmarks, is made of the fundus. Several examinations should be made with the patient in different postures—sitting, supine, lateral, and even prone; of these the supine is most important, since this is the position in which the operation is usually performed. Changes in posture may reveal a retinal tear which has hitherto been hidden by a retinal fold. Accurate localisation of the retinal tear or holes in relation to the outside of the sclera is essential; it is done

by assessing in terms of the clock-face the meridian in which the hole lies. Its distance from the ora serrata is judged ophthalmoscopically in terms of optic disc diameters (= 1·5 mm.). The patient rests in bed two or three days before operation.

The operation consists in reflecting a flap of conjunctiva and Tenon's capsule, and if necessary dividing an extraocular muscle over the site of the retinal hole (Fig. 212). An application of surface diathermy with a 3 mm. diameter blunt terminal is made on the sclera, using a current of 80 milliamperes for seven seconds. This causes coagulation in the

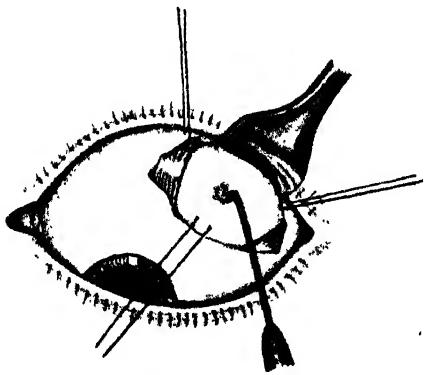


FIG. 212.—Perforating diathermy through the site of superficial diathermy.

choroid, which should be confirmed by immediate ophthalmoscopic examination. It appears as a white patch of exudate about 4 mm. in diameter. Its relationship to the retinal hole determines the sites of further applications of surface diathermy, each being checked by ophthalmoscopic examination. The object is to promote adhesion of the retina and choroid around the hole after the subretinal fluid has been evacuated. After satisfactory circumvallation of the hole by surface diathermy perforations of the sclera and choroid are made by a diathermy needle carrying a current of 40 milliamperes for three seconds. The subretinal fluid seeps out, and its evacuation is completed by suction. Ophthalmoscopic examination should then show apposition of the retina to the choroid. Both eyes are bandaged, and the head is immobilised

in a position such that the site of the hole is the most dependent part.

In favourable cases adhesion between retina and choroid is fairly firm in three or four weeks, but great care must be taken not to jeopardise its security by undue physical exertion.

Tumours of the Retina. See p. 428.

Cysts of the Retina are commonly found in the microscopic examination of degenerated eyes, especially near the ora serrata in old people ; they may lead to holes, disinsertion, and detached retina. Larger cysts occur elsewhere, and are sometimes due to adhesion of folds of detached retina. In rare cases large cysts may simulate detached retina clinically.

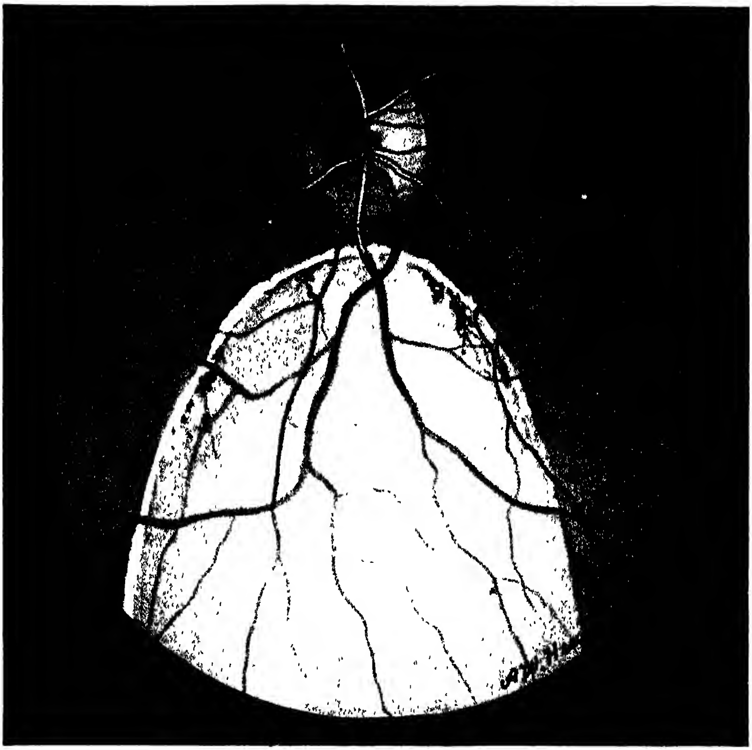
Congenital Abnormalities of the Choroid and Retina

Coloboma of the Choroid and Retina is a congenital malformation in which the choroid and retina are more or less badly developed over a certain area, usually the lower part (typical coloboma). The typical coloboma is due to defective closure of the fœtal, so-called choroidal, cleft. Ophthalmoscopically there is a glistening white area, usually with patches of pigment at the edges (Plate XVI.). There is often also coloboma of the iris (*q.v.*), and the eye may be small (microphthalmia). The condition is often bilateral, and is frequently hereditary. The patch is oval or comet-shaped with the rounded apex towards the disc, which may be included or not. A few vessels are seen over the surface, some retinal, others derived from the choroid at the edges, but most derived from the posterior ciliaries directly. The surface is often depressed irregularly (ectatic coloboma). The central vision is generally bad, and there is a scotoma in the field corresponding more or less accurately with the coloboma, though this usually contains some retinal elements near the edges.

Similar patches, often symmetrical in the two eyes, occur in other situations (atypical coloboma), notably at the macula (central or macular coloboma). It is probable that some of these are due to intra-uterine inflammation, particularly toxoplasmosis (*q.v.*).

Albinism is the defective development of pigment throughout the body. Owing to absence of pigment in the eye the iris looks pink, and the patients suffer much from dazzling. Nystagmus, photophobia, and defective vision—partly due to myopia, or, less frequently, hypermetropia—are present.

PLATE XVI.



Coloboma of the choroid.

[To face p. 384.

PLATE XVII.

Opaque nerve fibres.

There may be strabismus, usually convergent. The condition is hereditary. With the ophthalmoscope the retinal and choroidal vessels are seen with great clearness, separated by glistening white spaces where the sclerotic shines through. Microscopic examination has shown that total albinism is extremely rare, as traces of pigment have always been found in the retinal epithelium.

Partial albinism is commoner, and the absence of pigment is then limited to the choroid and retina, the irides being blue. There may be pigment in the macular regions, which may therefore look normal. People with dark hair sometimes have relatively slight pigmentation in the periphery of the fundus,

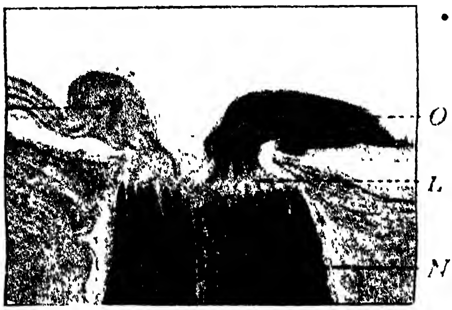


FIG. 213.—Opaque nerve fibres (*O*), stained by the Weigert-Pal method. *R*, retina. *L*, lamina cribrosa. *N*, optic nerve.

so that the choroidal vessels are seen: these patients will always be found to have had very fair hair as children.

Treatment consists in correction of the refraction by glasses, which should be tinted.

Congenital Pigmentation of the Retina. Small oval grey spots or groups of polygonal greyish-black spots are occasionally met with in the retina in routine examination of the fundus. They are flat and lie below the vessels, and remain unchanged indefinitely. They are probably congenital and due to heaps of retinal pigment epithelium similar to those forming melanomata in the iris (*q.v.*). (See also Nævus of the Choroid, p. 426).

Opaque Nerve Fibres. The medullary sheaths of the fibres of the optic nerve cease normally at the lamina cribrosa. Occasionally patches of fibres regain these sheaths after they have passed through the lamina cribrosa (Fig. 213). They appear ophthalmoscopically as white patches, the peripheral

edges of which are radially striated, looking as if frayed out (Plate XVII.). Usually the patches are continuous with the disc; occasionally they are isolated, but rarely far from the disc. Usually the retinal vessels are covered in places by the opaque fibres. When present the blind spot is enlarged, or a scotoma corresponds with the position of the patch. Very rarely the patch is large and involves the macula, so that central vision is abolished. If glaucoma or optic atrophy causes the fibres to degenerate the medullary sheaths disappear and no trace of the abnormality remains. It is important to be able to diagnose them, since they may be easily mistaken for exudates, *e.g.*, albuminuric retinitis. They not infrequently occur in both eyes. They are not strictly speaking congenital, for myelination of the optic nerve progresses from the brain towards the periphery, and is not completed until shortly after birth.

CHAPTER XVIII

Diseases of the Optic Nerve

THE optic nerve may be attacked by inflammation at any part of its course. The head of the nerve within the globe is frequently affected alone, and this condition is often called optic neuritis or papillitis. When the nerve is affected behind the eye the condition is called retro-bulbar or retro-ocular neuritis.

Hyperæmia of the Optic Disc is a condition which can rarely be diagnosed with certainty. Perfectly normal discs of different individuals show variations in colouring, and slight differences of illumination alter the appearances. There is no doubt that hyperæmia occurs as a precursor of optic neuritis, and in some of these cases it is possible to distinguish greater redness of the disc in one eye than in its fellow under the same conditions of examination.

Papillitis (*Intraocular Optic Neuritis*) **and Papillœdema.** Optic neuritis or papillitis is a term often applied to two groups of cases which should be carefully distinguished, viz., as part of a neuro-retinitis (*vide* pp. 350, 366), and as a result of intracranial disease. The pathology of the two conditions is different, though the ophthalmoscopic features may be almost or quite identical. In both there is œdema of the nerve-head, which is associated with true inflammatory changes in papillitis, and is passive in papillœdema. The general features will be considered first, the differences being discussed afterwards.

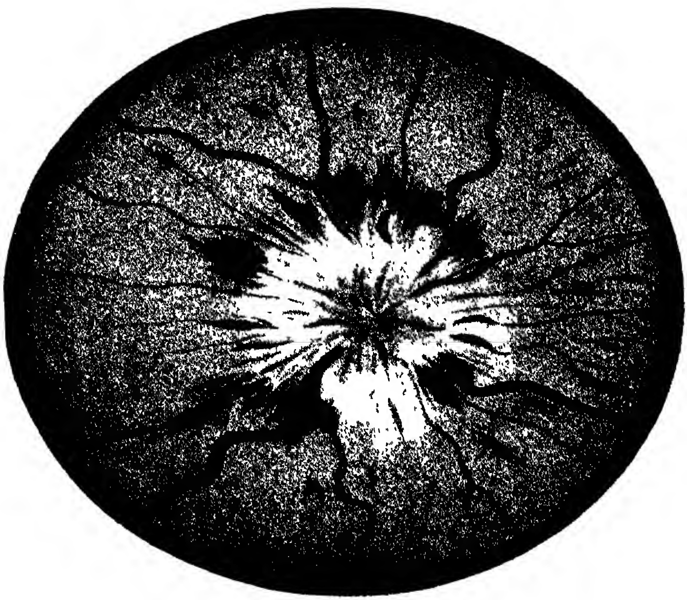
It has already been pointed out that the colour of the disc is a fallacious criterion of abnormality. Attention should be directed especially to the edges, which will always be found blurred, usually first on the nasal side, later in the whole circumference. The blood vessels are altered, the arteries being small, the veins distended. In the early stages the disc is usually redder than normal. In the later stages the blurring of the edges is much greater, and the disc looks larger than usual. Exudates cover the vessels in places, and the veins are greatly distended and very tortuous. The papilla is paler than normal and may be white; it shows radial streaks, and small hæmorrhages are generally present

upon it and the surrounding retina. The disc is now quite definitely and measurably swollen (*vide* p. 121).

In the papillitis of neuro-retinitis the swelling of the disc is usually moderate—2 or 3 D—shelving off gradually into the surrounding retina, which shows the signs of retinitis (*vide* p. 349). The disc is redder than normal, owing to dilatation of the capillaries, but the distension and tortuosity of the veins are moderate. The "optic neuritis" of intracranial disease may in some cases be a true descending neuritis, and will then show the condition just described, except that there is little or no retinitis. The intracranial disease in these cases is usually of an inflammatory nature, *e.g.*, meningitis. More commonly, however, the condition is one of intense œdema with no true inflammation, and this gives rise to papillœdema or "choked disc" (Plate XVIII., Fig. 1). Here there is very great swelling—up to 8 or 10 D—usually delimited much more definitely from the surrounding retina, which shows little change. The veins are enormously distended and very tortuous; the vessels are hidden in places by white exudates. There are frequently hæmorrhages on the swollen papilla and at its edges. In many cases it is impossible to distinguish ophthalmoscopically between the two forms. The swelling may be unusually great in neuro-retinitis, and *per contra* the appearances due to intracranial disease may simulate the neuro-retinitis of renal disease, including the star figure at the macula, particularly in children. The star figure is rarely complete in these cases; usually it is a fan-shaped figure on the disc side of the macula. It occurs only in severe cases, and is therefore commonest with cerebellar tumours. It may disappear completely after decompression, leaving an apparently normal macula.

The symptoms may be extremely vague. Central vision may be quite normal, in which case the pupils will also be normal in size and reactions. It is very important to remember this fact, which emphasises the necessity of examining the fundus in all cases of headache, &c. Even in this stage there may be some concentric contraction of the field of vision. Transient attacks of blurred vision, lasting for a few minutes up to an hour or so, are not uncommon in the early stages of papillœdema. Visual acuity may be practically unaltered throughout. It is usually diminished, but the loss bears no direct relationship to the amount of swelling of the disc. Later central vision is reduced, even to complete blindness. The pupils will then be large and immobile. In less severe cases or

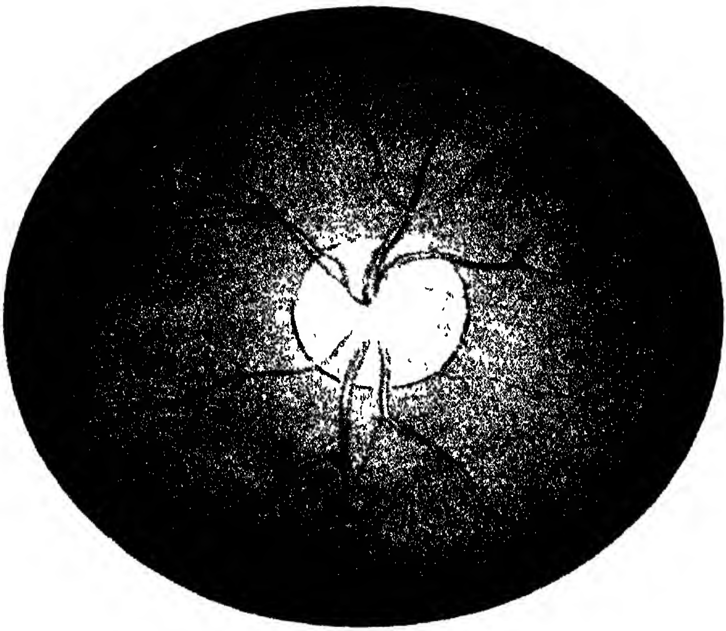
PLATE XVIII.



Papilloedema of intracranial disease.

[To face p. 388.

PLATE XIX.



Primary optic atrophy.

in the intermediate stage central vision is defective, the field is markedly reduced concentrically, and relative scotomata—first to green and red—or absolute scotomata may be present. There may be hemianopia or other defects in the field dependent upon the site of intracranial lesion. When there is extreme loss without much papillœdema it is probably due to the distended third ventricle pressing upon the chiasma and optic tracts. It is noteworthy in this relation that the diminution of vision is less with temporo-sphenoidal tumours than with those situated elsewhere (Paton). Vision may be normal in spite of a macular fan. Premonitory attacks of blurred vision are commonest with cerebellar tumours, possibly owing to interference with the circulation in the occipital lobes. Positive subjective phenomena, *e.g.*, seeing coloured lights, &c., are rare. In the papillitis of neuro-retinitis central vision is always reduced.

Both in neuro-retinitis and in intracranial disease the condition is generally bilateral, though not necessarily equal on the two sides. The relative amount of swelling may be of localising value in the case of intracranial disease, but its value has certainly been over-estimated; in frontal tumours and middle ear disease the swelling is usually greater on the side of the lesion. The time of onset of the papillœdema is really more to be taken into account than the amount of swelling, the localising value being attached to the side first affected. Thus the swelling may be actually less on the side first affected owing to subsidence associated with commencing atrophy. Unilateral papillœdema, with or without "secondary" optic atrophy on the other side, suggests a tumour of the opposite olfactory lobe or orbital surface of the frontal lobe or of the pituitary body (*vide* p. 393). Unilateral papillœdema occurs in the early stages of increased intracranial pressure and in orbital diseases such as tumours of the optic nerve or orbit, cellulitis of the orbit, hæmorrhage into the sheath of the optic nerve, &c.

The course is chronic, the prognosis bad. Occasionally, especially in syphilis, the disease subsides under treatment and good vision is preserved. The same applies to cases due to intracranial pressure if the pressure is relieved early. Palliative decompression by trephining the skull has a remarkable effect. Headache, vomiting and stupor are relieved, vision improves rapidly unless the nerves have been irretrievably damaged, and the papillœdema quickly subsides. Rarely the discs regain a normal appearance, but often the nerve fibres are destroyed, "post-neuritic" atrophy (*q.v.*) follows, and the patient may become blind

(*vide* p. 394). Recurrence of papillœdema is very rare, but has been recorded.

Diagnosis is easy in severe cases ; it may be very difficult in slight cases. Here the colour of the disc is no sure guide unless there is undoubted difference between the two eyes. Attention should be directed to the edges of the disc ; if these can be seen clearly defined with any lens there is no neuritis, but it does

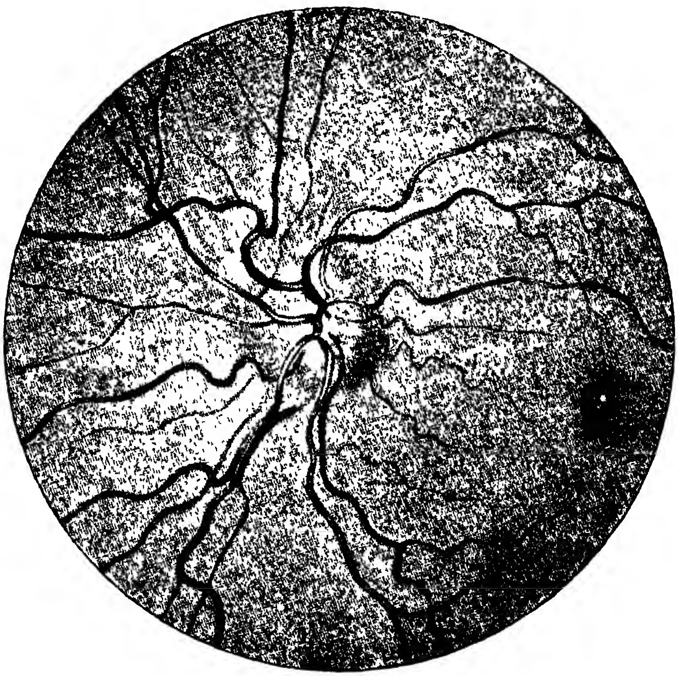


FIG. 214.—Pseudo-neuritis.

not follow that there is neuritis if they appear blurred. Astigmatism causes apparent blurring of the disc margin and there is a condition nearly simulating slight neuritis known as pseudoneuritis, seen particularly in hypermetropic eyes and due largely to a peculiar reflex (Fig. 214). In such cases attention must next be directed to the amount of swelling. In the absence of other indubitable signs such as exudates or hæmorrhages papillitis or papillœdema should not be diagnosed unless at least 2 D of swelling can be demonstrated. In some cases it is

necessary to keep the patient under careful observation for a considerable period before certainty can be arrived at.

The following stages may be distinguished in the development of papillœdema (Marcus Gunn, de Schweinitz and Holloway) :—

(1) Increased redness of the disc, with blurring of its upper and lower margins, with a gradual progression of the blurring to the nasal edges, while the temporal margin is still visible, represents the first stage.

(2) Increased œdema of the nerve-head, beginning filling in of the physiological pit, involvement of the temporal margin of the disc, with a tendency of the œdema to spread into the surrounding retinal area, and uneven distension and darkening of the retinal veins, represent the second stage.

(3) Decided increase of œdema, elevation and size of the nerve-head, with vascular striation of the swollen tissue and striæ of œdema in the form of lines in the swollen retina between the disc and macula, marked distension of the retinal veins and retinal hæmorrhages, represent the third stage.

(4) Increase in the prominence of the disc, which assumes a mound-shape and begins to lose its reddish and juicy colour and to become opaque, exudation in and on the swollen disc and surrounding retina, elaboration of the retinal hæmorrhages in size and number, represent the fourth stage.

(5) Decided subsidence of the vascularity of the papillœdema and increasing pallor, with or without sinking of its prominence, apparent contraction of the retinal arteries and thickening of their perivascular lymph-sheaths, spots of degeneration of the retina, especially in the macula, represent the fifth stage, which passes into the final stage of so-called post-neuritic atrophy.

Ætiology. The chief causes of papillitis are those of neuroretinitis (*q.v.*) and of papillœdema intracranial disease. The latter is the more frequent: at least 80 per cent. of cases of intracranial tumour give rise to papillœdema. Any intracranial tumour in any position, with the exception of the medulla oblongata, may cause papillœdema, the highest percentage being found with tumours of the mid-brain, parieto-occipital region, and cerebellum (*vide* p. 607). The papillœdema is independent of the nature of the tumour and of its rate of growth. The size of the growth is important only in relation to its site. It has been said that papillœdema is less likely to occur in myopic eyes, but this is erroneous. Meningitis is the next commonest cause, especially tuberculous meningitis—basal meningitis relatively rarely. Other intracranial causes are abscess (*vide* p. 608), thrombosis of the cavernous sinus, aneurysm, hydrocephalus (rarely), &c.

Toxæmia accounts for most of the other cases of "optic neuritis." Syphilis may act in this manner as a basal meningitis, or more frequently as an intracranial gumma; it is a frequent cause. Papillitis may be due to any of the acute febrile diseases, but only in exceptional cases, and to acute anæmia from sudden loss of blood. It occurs sometimes with poisoning with lead and other substances which usually give rise to toxic amblyopia of retro-bulbar type (*vide* p. 399). It has often been described in chlorosis, and suppression of menses has been given as a cause. This ætiology is in my

FIG. 215.—Papillœdema. (From a photograph by Coats.)

opinion doubtful; some cases have subsequently proved to be due to other more serious causes, and many are probably errors of diagnosis (*see* Pseudo-neuritis, p. 390). Slight papillitis has been observed in some of those rare cases in which there is persistent escape of cerebrospinal fluid from the nose. Malformation of the cranium, *e.g.*, acrocephaly, intra-orbital tumours and inflammations—caries, periostitis, &c. —and tumours of the optic nerve act directly upon the nerve.

Pathology. In neuro-retinitis there is true inflammation of the nerve, but even then œdema plays a prominent part owing to the obstruction to the outflow of venous blood at the site of the lamina cribrosa (*vide* p. 126). Most cases of "choked

disc " are at first caused by simple œdema, without inflammation; in the later stages there may be some inflammatory reaction—infiltration with leucocytes, &c.—due to the irritation of the necrosed tissues. The œdema occurs first on the lamina cribrosa and peripheral parts of the nerve; the physiological cup then becomes filled in and the internal limiting membrane raised (Fig. 215). The macular fan is caused by œdema in the nerve fibre layer and raising of the internal limiting membrane in folds; the outer reticular layer may be œdematous, but there are no large cystic spaces as in albuminuric retinitis. There is often sub-pial œdema distal to the site of entry of the central vessels into the nerve, but the nerve is normal proximal to this point. It is noteworthy that the central vein is collapsed where it crosses the sub-dural and sub-arachnoid spaces (*vide infra*). In many cases the sub-arachnoid space is so distended that it is ampulliform just behind the globe. The nerve-fibres become swollen and varicose, ultimately degenerating; they show cell-like bodies (cytoid bodies) as in albuminuric retinitis; these are not found behind the lamina cribrosa. The neuroglia proliferates and the mesoblastic tissue around the vessels becomes thickened. In a minority of cases due to intracranial disease, especially meningitis, there may be a descending neuritis, with true inflammation of the nerve.

The mechanism whereby œdema is induced by intracranial disease has been the subject of much dispute and is still an unsolved problem. There is no doubt that the predominant factor is increased intracranial pressure. This is proved by the fact that the œdema almost invariably subsides, even though the intracranial disease continues, if the pressure is relieved by freely opening the skull. If communication is cut off from one intravaginal space by pressure of a tumour of the pituitary body or olfactory region papillœdema does not occur on this side, but the optic nerve passes into a condition of partial or complete " secondary " atrophy (*vide* p. 404); this condition of papillœdema on one side and optic atrophy on the other is known as the Foster Kennedy syndrome, though it was described by Gowers. The following theories have been advanced to account for papillœdema: (1) that it is purely inflammatory; this is negatived by the histological findings; (2) disturbance of vaso-motor innervation: there is no evidence in favour of this view; (3) arterial anæmia, leading to altered conditions of osmosis at the nerve-head; (4) lymph stasis, owing to impediment to return of lymph *viâ* the

intravaginal space; (5) propulsion of cerebro-spinal fluid through the lamina cribrosa; (6) compression of blood vessels and local vascular engorgement either in the lamina cribrosa and nerve or in the vaginal space. It is most probable that papillœdema is due to compression of the central vessels as they cross the vaginal space, causing collapse of the vein, whilst the thicker walled artery continues to transmit blood.

The *treatment* of papillœdema is essentially that of the underlying cause. Local treatment is of no avail, but all sources of irritation, such as bright light, &c., should be guarded against. Intracranial pressure should be relieved before vision is lost, even if the cause is a tumour which cannot be completely extirpated.

If the localising symptoms are positive trephining should be performed over the site of the tumour if possible. If this is impossible or if localising signs are masked by pressure symptoms a free opening should be made in the skull supra- or subtentorially according to the indications of the case. The relief of pressure will alleviate the cerebral œdema and unmask the localising signs, so that it may be possible at a later stage to undertake a radical operation. Paralysis of the external recti is often due to the pressure on the sixth nerves in their long intracranial course, and in these cases is of no localising value (*vide* p. 564).

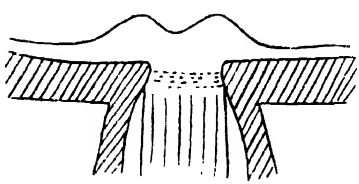


FIG. 216.—Diagrammatic meridional section of optic disc in papillœdema.

If decompression is done early the prognosis as to sight is very favourable. After decompression vision usually improves rapidly, but only if the intracranial pressure is effectually relieved. It is not sufficient merely to open the cranium; the dura mater must be incised. The recovery of vision may be much more rapid than the subsidence of the papillœdema. Vision may deteriorate after operation, probably owing to excessive sclerosis and proliferation in the disc. If delayed until there is great swelling and exudation, with marked depreciation of vision, and especially if signs of subsidence and commencing atrophy are present, further diminution of vision is to be anticipated. Subsidence of the papillœdema is usually rapid after operation, a decided change being seen in a week to a fortnight, though there is considerable variation in different cases. In cerebral abscess there may be temporary

increase of the swelling after operation, without, however, seriously compromising the prognosis.

The treatment of papillitis is that of the ætiological factor.

Retrobulbar Neuritis. The intra-orbital portion of the optic nerve may become inflamed as the result of extension from the surrounding tissues. Retrobulbar neuritis is usually divided into an acute, so-called symptomatic form, and a chronic, idiopathic form. The latter is the condition which is described as toxic amblyopia and which may be due to a primary retinal lesion.

Acute retrobulbar neuritis is usually unilateral. The patient complains of sudden obscuration of vision, which increases rapidly during one to eight days; there is sometimes pain on moving the eye. The pain is increased by pressure upon the globe, and neuralgia and headache may be present. The tenderness of the eyeball to digital pressure is limited to a small area corresponding roughly to the site of attachment of the superior rectus tendon. This sign is present only in the early stages of the disease and disappears in a few days (Greeves). Ophthalmoscopic examination will probably reveal a quite normal fundus. It is therefore very easy to overlook the true condition and to attribute the symptoms to hysteria. Careful methodical examination will minimise this danger. Diagnosis at this stage will depend upon thorough investigation of the pupil reactions and of the field of vision. The patient should be asked if he ever squinted (*vide* p. 414).

At first glance the pupil reactions will be apparently normal, both directly and consensually to light, as well as to accommodation. More minute inspection will show, however, that though the pupil of the affected eye reacts to light the contraction is not maintained under the bright illumination, *i.e.*, instead of remaining contracted the pupil slowly dilates while the light is still kept from the eye. *Lack of sustained constriction of the pupil to light,* if it can be placed beyond dispute, *is of the greatest diagnostic significance.*

The field of vision shows a central scotoma, which may be relative for colours or absolute. It is not always quite central, but may be paracentral or sectorial or in the form of a ring around the fixation point. There is usually some peripheral loss of the field and there may be complete blindness.

In the later stages or more severe cases there are usually

ophthalmoscopic changes. These are distension of the veins, with diminished calibre of the arteries, or actual papillitis, moderate in degree. They are most likely to occur if the focus is close behind the eyeball. With or without these preliminary changes atrophy of the optic disc may ensue. In every case in which the inflammation in the nerve behind the globe is so great as to lead to destruction of the nerve fibres in this situation the degeneration extends not only towards the brain but also towards the eye. In the milder cases pallor of the disc is limited to the temporal side, corresponding with degeneration of the papillo-macular fibres. It has already been noted that the ganglion cells at the macula are more liable to be affected by deleterious agents than those in other parts of the retina. The same fact applies to their axis cylinder processes, contained in the papillo-macular bundle, wherever in their course the noxious agent acts. This accounts for the clinical similarity between true retrobulbar neuritis and toxic amblyopia.

The causes of acute retrobulbar neuritis are local and general. Among the former periostitis and transmission of inflammation from the ethmoidal and sphenoidal sinuses may be mentioned, though this cause has certainly been exaggerated; attention, however, should be directed to any nasal complication. Hæmorrhage into the optic nerve sheath or orbit and fracture of the base of the skull, involving the optic foramen, may produce a similar clinical picture. Pressure on the chiasma by hypertrophy or tumour of the pituitary body or tumour of the frontal lobe may in the early stages cause the symptoms of a unilateral retrobulbar neuritis (*vide* p. 411: see also hereditary optic neuritis, *infra*). Among general diseases which cause retrobulbar neuritis insular sclerosis is the most important; it is a very frequent early symptom in this disease, which should always be suspected, especially when the symptoms occur in young women. Considerable recovery of vision usually occurs (*vide* p. 404), but recurrence is not uncommon (*vide* p. 597). Other alleged causes are rheumatism, chills, diabetes, infectious diseases, septic foci somewhere in the body (mouth, intestinal tract, &c.), and so on. In cases due to such causes relapses are common, and both eyes may be affected, together or alternately. Acute retrobulbar neuritis may be preceded by peripheral facial palsy of the same or opposite side, and shows some analogies to this condition (Marcus Gunn). The prognosis depends upon the cause and the possibility of combating it, but is generally good.

In multiple sclerosis the affection of the nerve never leads to complete blindness (*cf.* Tabetic Optic Atrophy, p. 403).

Treatment consists in attacking the cause, the mouth and nasal sinuses being specially carefully investigated. The eyes must be protected from bright light, and kept at rest by atropine and abandonment of near work. When the cause is obscure, mercury, iodides, salicylates, diaphoresis, and tonics may be used. Intravenous injection of vaso-dilators has been advocated (*vide* p. 398). Smoking should be prohibited. Cases in which disseminated sclerosis is suspected should be referred to a neurologist for further investigation. In diabetic cases appropriate régime should be instituted.

Toxic Amblyopia. Tobacco, alcohol, quinine, filix mas, carbon disulphide, stramonium, cannabis indica and other poisons sometimes produce defective vision, which is then known as toxic amblyopia. Tobacco and alcohol amblyopias, which are most frequently met with, are usually described as forms of retrobulbar neuritis (*q.v.*), but experimental and pathological evidence tends to show that the condition is primarily retinal in these cases.

Tobacco amblyopia results from the excessive use of tobacco, either by smoking or chewing; and also occasionally from the absorption of dust in tobacco factories. Smokers of shag and strong tobacco mixtures suffer most. In many cases there is also over-indulgence in alcohol. It is known that alcohol alone may produce toxic amblyopia, and cases in which tobacco caused the disease in total abstainers from alcohol have been published: indeed the visual loss in these cases is worse than in moderate drinkers. The patients may have smoked excessively for years with impunity, the attack coinciding with some intercurrent cause of debility, digestive disturbance, &c. They are usually thirty-five to fifty years of age.

The patient complains of increasing fogginess of vision, which is usually least marked in the evening and in a dull light. Central vision is greatly diminished. The field of vision is found to be full, but there is a central colour scotoma for red and green. This is usually small, horizontally oval between the fixation and blind spots, but in rare cases may extend to the limits of the red field, or even be absolute. In such cases the possibility of the presence of congenital colour-blindness should be borne in mind. Both eyes are about

equally affected. Ophthalmoscopically there may be congestive haze of the edges of the optic disc followed by undue pallor of the temporal side of the disc, but the changes are usually slight.

The prognosis is good if the toxic agents are discontinued absolutely, but improvement is slow, taking several months. The alcoholic cases in non-smokers are usually less severe and clear up rapidly. Recurrence is very rarely seen. Very rarely optic atrophy may result, but it is doubtful if these are uncomplicated cases.

The disease is probably due to poisoning of the ganglion cells of the retina. In experimental cases, and in one case examined in man, the cells showed vacuolation and breaking up of the Nissl granules. This leads to degeneration of the nerve fibres, demonstrable only after they have obtained their medullary sheaths, *i.e.*, behind the lamina cribrosa. The degeneration is found to be limited to the papillo-macular bundle (*vide* p. 75). The ganglion cells of the fovea and macular region are the most highly differentiated and are liable to suffer first and most severely in any toxic condition. The degeneration is therefore a wedge-shaped area on the temporal side of the nerve immediately behind the globe, but becomes a circular central area more posteriorly (Fig. 60). This degeneration was discovered early in the history of the disease, and combined with the clinical similarity to undoubted cases of retrobulbar neuritis led to the conclusion that it was the primary seat of the disease. Nicotine is generally regarded as the toxic agent, but it is much more probable that it is one of the more volatile decomposition products of nicotine, *e.g.*, collidine or lutidine.

The amblyopia produced by diabetes, carbon disulphide, and iodoform resembles that of tobacco. Diabetics appear to be specially susceptible to tobacco.

Treatment consists in total abstinence from tobacco and alcohol. It should be combined with tonic treatment, especially the administration of strychnine. Iodide of potassium may be given, and copious draughts of water, combined with exercise, have been recommended. Intravenous injections of vasodilatators, such as sodium nitrite (40–50 mgrm.), or erythrol tetranitrate (1·5 grain) by the mouth, are said to cause rapid improvement, and local injections of acetylcholine (*vide* p. 356) may also be tried. All patients with central scotomata, other than those due to total macular degeneration, should abstain from tobacco and alcohol.

Quinine amblyopia differs in some striking characteristics from tobacco amblyopia. Here total blindness (amaurosis) follows the use of the drug, even in such small doses as 12 grains in susceptible persons; 40 grains is the maximum amount of sulphate of quinine which should be given within twenty-four hours (Yarr). The largest doses are usually taken for malaria, but quinine is also used as an abortifacient. The pupils are dilated and immobile. Deafness and tinnitus aurium are present. Ophthalmoscopically the retinal vessels are extremely contracted and the disc is very pale; œdema of the retina has been described in the early stage. In less marked cases or at a later stage the fields of vision are much contracted. The fields gradually widen out, but do not regain their normal limits. Central vision may be completely restored. The discs may remain pale for years or become normal. Occasionally blindness is permanent and optic atrophy ensues. The same condition may follow administration of ethyl-hydrocuprein (optochin) for pneumonia, and from excessive doses of dial and other barbituric compounds, but in the latter cases some vision is regained if the patient survives.

Salicylic acid and *salicylates* occasionally produce an amblyopia of the same type and with the same ophthalmoscopic features as that of quinine, but not so severe.

Treatment consists in discontinuing the drug, administering amyl nitrite or nitro-glycerine, supplemented by strychnine and digitalis, or local acetylcholine injections.

The amblyopias produced by *methyl alcohol, arsenic, lead, nitro-* and *dinitro-benzol*, and *filix mas* differ from those of retrobulbar type in the more serious optic atrophy which generally ensues. There is probably always a stage at which a central scotoma is present, but it is often missed.

Methyl alcohol poisoning from drinking wood-alcohol was common in America during prohibition, and used to occur in England from drinking methylated spirit before it was intentionally adulterated. Nausea, headache, giddiness, &c., are followed by coma. If the patient survives, vision very rapidly fails, passing through the stage of contracted fields and absolute central scotoma to blindness. Vision may improve, but usually again relapses, becoming gradually abolished by progressive optic atrophy. Rarely restoration is complete. Ophthalmoscopically there may be blurring of the edges of the discs and diminished size of the vessels, in the early stages. Later there are signs of optic atrophy, usually of the primary type (*vide* p. 401).

There is widespread degeneration of the ganglion cells of the retina.

Arsenic is specially liable to cause optic atrophy, usually total, when administered in the form of pentavalent benzol-ring compounds such as atoxyl or soamin, arsacetin, hectine, &c. These were used for attacking the trypanosome of sleeping sickness, but have now been abandoned. The salvarsan group have the arsenic in trivalent combination and are less toxic: no cases of optic atrophy have been reported from their use.

Lead poisoning is rarely seen since precautions have been taken to eliminate salts of the metal from pottery glazes, &c. The ocular signs are optic neuritis or optic atrophy, which may be primary or post-neuritic. Some cases have retinitis, which may be due directly to lead or of albuminuric type, secondary to lead nephritis

Filix mas, used as a helminthetic, may cause amblyopia in excessive doses, especially if given with castor oil. The ophthalmoscopic picture is said to resemble that of quinine amblyopia. Later, optic atrophy supervenes. I have seen a case in which a drachm of extract of male fern was ordered three times a day and was taken for ten days. There was total optic atrophy in one eye and partial atrophy with much contraction of the field in the other.

Hereditary Optic Neuritis (*Syns.—Hereditary Optic Atrophy, Leber's Disease*) is a form of retrobulbar neuritis, usually commencing at about the twentieth year of life. Descent is generally through an unaffected female to the males, though females are also sometimes affected. Vision generally fails rapidly at first, then gradually, but remains stationary or gradually improves after six months. Both eyes are always affected, though one may precede the other by a few days up to eighteen months. In two-thirds of the cases there is a central scotoma, either partial for colours or also for white. The peripheral field is usually normal, but concentric contraction or sector-shaped defects may occur. Total and permanent colour-blindness has been known to follow. The central scotoma generally persists, but progressive constriction of the field to complete blindness is rare. Members of the same family often show identical peculiarities in the progress of the cases. The fundus is at first normal or there is slight blurring of the edges of the disc. In later stages, after months, optic atrophy ensues, with pallor confined to the temporal side or involving the whole disc. Apart from headache, migraine, &c., the general health is good. Fisher has suggested that Leber's disease is due to transitory changes in the pituitary body (*vide* p. 413), resulting in pressure upon the chiasma and associated with the periods of physiological change in the sexual life. If this be true treatment with thyroid and pituitary extracts may be good.

Optic Atrophy is the term usually applied to the condition of the disc when the optic nerve is degenerated. It has been pointed out that injury to the nerve fibres in any part of their course from the retina to the external geniculate body leads to degeneration not only on the proximal (cerebral) side—as might be anticipated for afferent fibres—but also on the distal (ocular) side (*vide* p. 396). Optic atrophy follows extensive disease of the retina from destruction of the ganglion cells, *e.g.*, in retinitis pigmentosa; it also follows destruction of the nerve in the orbit, as in injury of the nerve at the optic foramen in fracture of the base of the skull, severe retrobulbar neuritis, &c. The break in continuity of the fibres may be at the disc itself, such as results from the strangulation of the papillitis of neuro-retinitis or papillœdema. These cases are distinguished as "*post-neuritic*" *atrophy*, owing to special features which they often exhibit. Besides these causes there is a well-defined group of cases in which optic atrophy occurs without previous evidence of severe local inflammation, but associated with general disease, usually of the central nervous system, or without discoverable disease. Such cases are described as *primary atrophy*.

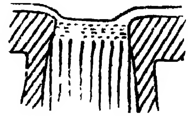


Fig. 217.—Diagrammatic meridiona section of the optic disc in atrophic cupping. Note that the lamina cribrosa is not displaced (*cf.* Fig. 162).

The essential ophthalmoscopic features of optic atrophy in general are alteration in the colour of the disc and changes in the blood vessels. The disc is always pale, but may show varieties of tint specially associated with various types of atrophy. The pallor affects the whole disc and must be carefully distinguished from the white centre, often encroaching upon the temporal side, due to physiological cupping. The pallor is not due to atrophy of the nerve fibres, but to loss of vascularity; hence it is an uncertain guide to visual capacity (*vide* p. 403). The change in the vessels is always on the side of contraction, but may be absent.

In primary (grey, tabetic, spinal) atrophy the disc is grey or white, sometimes with a greenish or bluish tint (Plate XIX., Fig. 1). The stippling of the lamina cribrosa is seen; the edges are sharply defined, and the surrounding retina looks normal. Owing to the degeneration of the nerve-fibres (Fig. 218) there is slight cupping (atrophic cupping) (Fig. 217), which must be carefully distinguished from glaucomatous cupping. It is shallow and saucer-shaped, as shown by the

slight bending of the vessels, but is scarcely measurable with the ophthalmoscope. There is no retraction of the lamina cribrosa. The vessels are normal or only slightly contracted. Both eyes are generally affected.

In the "secondary" atrophy of retrobulbar mischief the condition nearly resembles primary atrophy, but there is greater pallor, and the vessels are more likely to be contracted. In the "consecutive" atrophy of retinal and choroidal disease, as typically represented by the late stages of retinitis

Fig. 218.—Tabetic Optic Atrophy. (From a photograph by Coats.) The medullary sheaths of the normal nerve-fibres are stained black by the Weigert-Pal method.

pigmentosa, the disc has a yellowish waxy appearance, the edges are less sharply defined, and the vessels are very markedly contracted, sometimes to almost complete disappearance. This type of atrophy occurs in severe cases of disease of the retinal blood-vessels.

Post-neuritic atrophy may be indistinguishable from the other forms ophthalmoscopically. More commonly it can be diagnosed with considerable probability by characteristic signs (Plate XIX., Fig. 2). These depend upon the fact that the absorption of the exudates leads to a certain amount of reactionary organisation, with the formation of a variable

quantity of fibrous tissue upon the disc. This tissue obscures the lamina cribrosa and fills in the atrophic cup. It extends over the edges, which are thus indefinite, and along the vessels as a thickening of the perivascular sheaths. Further, it throttles the vessels, so that they become markedly contracted, especially the arteries. Owing to previous neuroretinitis the surrounding retina often shows permanent changes, chiefly manifested by pigmentary disturbance. When such changes are well marked previous papillitis may be hypothesized with great probability, but in their absence the conclusion that there has not been papillitis is not justifiable. The amount of reactionary organisation varies greatly in different cases, and the tissue laid down is in the course of time gradually absorbed to some extent. It must also be remembered that the amount of fibrous tissue on the normal disc varies considerably and that slight filling in of the physiological cup with shreds of fibrous tissue extending outwards along the vessels occurs as a congenital peculiarity.

In total optic atrophy the pupils are dilated and immobile to light and the patient is blind. When unilateral the consensual reaction to light is exaggerated. In partial optic atrophy central vision is depressed and there is concentric contraction of the field, with or without scotomata, relative or absolute, according to the cause. In primary atrophy the disease is usually slowly progressive, ending after months or years in complete blindness. It is important to note that no deduction as to the amount of vision can be made from the ophthalmoscopic appearances. The presence of all the signs of atrophy is not inconsistent with a certain, sometimes considerable, amount of vision.

The chief cause of primary optic atrophy is tabes. It may be the first sign, and the other symptoms and signs may be long delayed. The patient should be carefully investigated for a history of syphilis, the presence of Argyll Robertson pupils, the absence of knee-jerks, the presence of anæsthesia (especially of the fifth nerve), inco-ordination, a positive Wassermann reaction, and so on. If optic atrophy occurs early in tabes, ataxy may be long delayed or never supervene. On the other hand, if it occurs late there is no appreciable difference in the course of the ataxy. Both eyes are affected, but often in unequal degree. The disease advances slowly but surely to complete blindness—progressive optic atrophy *par excellence*. The lesion was formerly supposed to be in the ganglion cells of the retina, but there is now considerable evidence that

the earliest change is an inflammatory exudation into the intracranial part of the nerve and the chiasma. A similar condition may be due to general paralysis and insular sclerosis, less commonly to other forms of central nervous disease; in insular sclerosis the course of the disease is usually different —variations, or repeated acute attacks, never causing complete blindness. The more favourable prognosis in disseminated sclerosis may be due to the fact that in this disease the axis cylinders often escape in spite of much destruction of their medullary sheaths. Primary atrophy may be due to poisons, such as methyl alcohol, lead, atoxyl, &c. (*vide* p. 399). It occurs rarely after repeated large hæmorrhages from the stomach, uterus or nose in unhealthy subjects; seldom, if ever, in healthy subjects, as, for example, after wounds in war.

In primary atrophy, though central vision is early affected, there is usually no central scotoma. Cases of tabes in which it is said to have occurred (Fuchs) may possibly be due to over-treatment with arsenical preparations. The field shows progressive concentric contraction, often with marked indentations which are rather more common on the nasal side. Finally, usually in a year or two in tabes, the fixation point is engulfed, though eccentric perception of light may still persist for a while on the temporal side. Contraction of the colour fields precedes that of the field for white, so that there is a stage of acquired colour-blindness. The prognosis is very bad.

"Secondary" atrophy is caused by compression of the optic nerves, chiasma, or tracts by tumours, aneurysm, distension of the lateral ventricles (hydrocephalus), &c. These tumours, &c., are so situated as to press directly upon the nerve fibres without causing much rise of intracranial pressure, as in the case of tumours of the pituitary body, aneurysm of the internal carotid in the cavernous sinus, &c., or to press directly on the chiasma or optic nerves behind the optic foramen, thus blocking communication with the intravaginal lymph-space, as in the case of tumours of the olfactory lobe and inferior aspect of the frontal lobe (*vide* p. 393). The same type of atrophy may follow injury to the optic nerve at the foramen, hæmorrhage into the dural sheath, section of the nerve, compression by blood clot, acrocephaly, &c., without previous neuritis. Injury to the optic nerve is often due to fracture of the base of the skull, and may be bilateral. In many cases the injury probably takes the form of a rupture of

the small pial vessels supplying the nerve. Vision in the eye and direct reaction of the pupil to light are immediately lost, but pallor of the disc does not supervene until the second or third week later.

The prognosis and treatment of post-neuritic atrophy have been dealt with in discussing papillœdema.

Treatment of primary atrophy is that of the cause. For the lesion of the nerve itself mercury, iodides, strychnine, nitroglycerine, the constant current, &c., may be used. Attempts have been made to destroy the spirochætes in the central nervous system in tabetic atrophy by raising the body temperature in various ways, *e.g.*, by induction of malaria; the deterioration of vision has been stopped in a number of cases by this means. Efforts to improve the local vascular supply by decompression operations have had no success.

Tumours of the Optic Nerve. See Chap. XXXIII.

Congenital Abnormalities of the Optic Disc

Coloboma of the Optic Disc. This occurs in two forms, one of which is common, the other rare. The common form is due to incomplete closure of the choroidal fissure, and manifests itself as an *inferior crescent,* much resembling the myopic crescent (*q.v.*), but situated at the lower edge of the disc (Plate VIII., Fig. 1). It is a crescent, whiter than the disc itself, situated at the lower border. It occurs most commonly in hypermetropic and astigmatic eyes, which are often found to have slightly defective vision in spite of correction of any error of refraction. It is often slightly ectatic (conus).

In what is commonly known as *coloboma of the disc* (or nerve sheath) there is greater failure of the fœtal fissure to close. The disc then looks very large and the vessels have a very abnormal distribution, appearing only above or irregularly round the edges. The apparent disc is really the sclerotic and inner surface of the sheath of the nerve, the nerve itself being usually spread out as a pink horizontal linear band at the upper part. The floor of the coloboma is white and measurably depressed, often quite ectatic. The eye usually has defective vision.

Rarer anomalies allied to coloboma are round "*holes*" in the disc, usually looking grey or black owing to the shadow, and situated in the temporal portion of the disc;

and patches of *pigment* due to inclusion of retinal pigment epithelium.

Mention has already been made (p. 403) of excess of *fibrous tissue on the disc* and extending a short distance along the vessels. Sometimes the fibrous tissue takes the form of a delicate semi-transparent membrane covering the disc and appearing to be slung from the vessels.

CHAPTER XIX

Symptomatic Disturbances of Vision

Apart from the disturbances of vision which have been already considered and have their origin in the eye itself, there are others dependent upon lesions in the visual nervous paths. Since they not infrequently closely simulate the disorders due to peripheral causes, or are early evidence of disease, they lead the patient to consult an ophthalmic surgeon. There are also visual defects the cause and seat of which are imperfectly elucidated ; though some are probably peripheral in origin, it will be convenient to consider them here.

Hemianopia (*Syns.— Hemianopsia, Hemiopia*). Hemianopia denotes loss of half of the field of vision. The commonest clinical form is so-called *homonymous hemianopia*, in which the right or left half of the binocular field of vision is lost, owing to loss of the temporal half of one field and the nasal half of the other. The condition may be due to a lesion situated in any part of the visual paths from the occipital lobe to the chiasma. A focus of disease in this area causes loss of vision of the corresponding halves of each retina (hence the designation homonymous), *i.e.*, loss of the opposite halves of the visual fields.

In many cases of hemianopia the fixation area in each field escapes (Fig. 219) especially if the lesion is near the occipital cortex; in infra-geniculate lesions the more usual occurrence is a bisection of the fixation point. In a number of cases it is probable that the macular fibres are in fact spared owing to their widespread but segregated course in the optic radiations and their separate representation in the occipital pole. The immunity of the macula in vascular lesions of the cortex is attributed to the fact that the occipital pole is supplied by the posterior and middle cerebral arteries both of which are seldom blocked at the same time. The explanation in other cases is not so obvious. The theory that there is a decussation of macular fibres either in the retina, chiasma or corpus callosum has not been substantiated. In certain cases the sparing of the macula may be only apparent owing to a functional shift of fixation towards the seeing part of the retina. On the other hand, a possible explanation may be

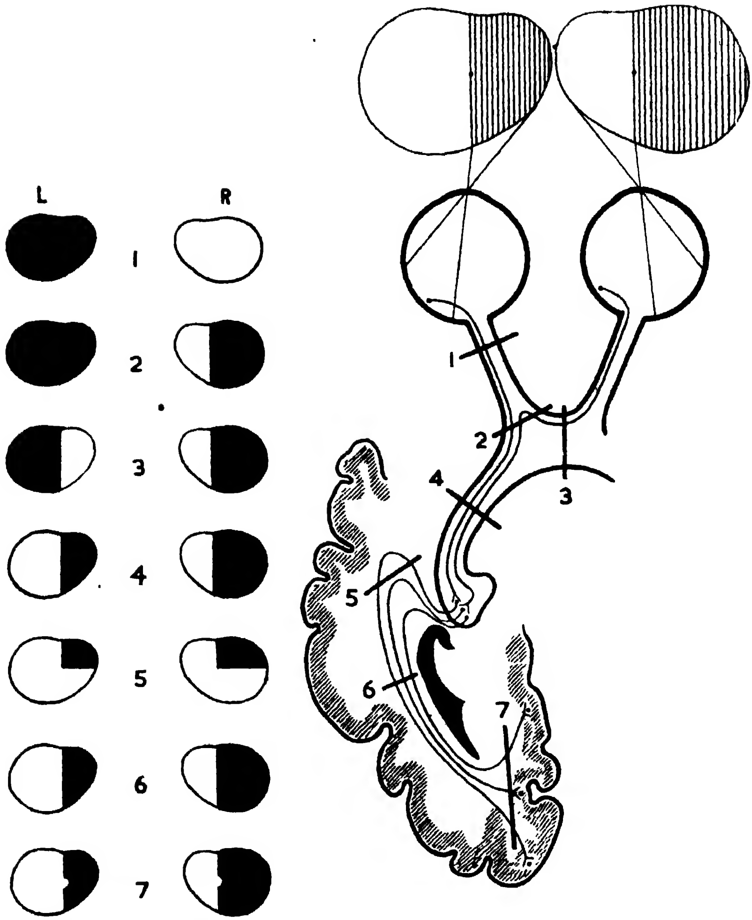


Fig. 219.—Diagram of the visual paths, showing sites of lesions and the corresponding field defects.

1. Lesion through optic nerve—ipsilateral blindness;
2. Lesion through proximate part of optic nerve—ipsilateral blindness with contralateral hemianopia;
3. Sagittal lesion of chiasma—bitemporal hemianopia;
4. Lesion of optic tract—homonymous hemianopia;
5. Lesion of temporal lobe—quadrantic homonymous defect;
6. Lesion of optic radiations—homonymous hemianopia sometimes sparing the macula);
7. Lesion of occipital lobe—homonymous hemianopia (usually sparing the macula).

sought in the integrative powers of the central visual mechanism (Verhoeff).

Lesions of the external geniculate body cause homonymous hemianopia (Fig. 219); those limited to the pulvinar and superior colliculus do not. Right hemianopia is much more quickly discovered than left, owing to the fact that reading is impossible. Left hemianopia is often discovered by the fact that the patient does not see food on the left side of the plate.

Cortical and Sub-cortical Lesions. The majority of cases of hemianopia are due to lesions above the primary visual centres, usually in the occipital lobe or optic radiations (Fig. 219, 6). The injury or disease rarely affects the grey matter of the occipital cortex only; the sub-cortical white matter is almost invariably involved. The chief causes are injury by falls on the back of the head or gun-shot wounds, cerebral tumour, cerebral softening due to syphilitic or other disease of the blood vessels, &c. In gun-shot wounds both occipital lobes are not infrequently injured. There is usually unconsciousness from concussion at first; with the gradual recovery the hemianopia becomes manifest. If both lobes are extensively injured there is bilateral hemianopia with complete blindness. Often, however, some portion of the cortex of one or other calcarine fissure escapes, and in these cases some measure of central vision is regained. In less extensive injury the hemianopic symptoms may gradually improve. The first sign of improvement is the perception of the movement of objects in the affected field, the nature and details of the objects being as yet quite unrecognised. The onset of hemianopia due to disease of the cortex is more gradual, and careful investigation with the perimeter shows that the colour fields are often lost before the field for white light, but the field for white is always contracted in these cases (Gordon Holmes). This *hemiachromatopsia* is itself of gradual onset, the colour fields becoming contracted. In cortical and sub-cortical lesions the pupil reactions are normal (*vide* p. 56), and in many cases the fundi reveal no ophthalmoscopic changes. The chief exception to the latter statement is in the case of tumours of the occipital lobe, in which case the rise of intracranial pressure leads to bilateral papillœdema. Cortical lesions are liable to be accompanied by word-blindness, probably due to involvement of the angular gyrus. When the lesion is in the posterior part of the internal capsule hemianæsthesia, with or without hemiplegia, is likely to be present.

Rare cases of homonymous *quadrant hemianopia* have been reported, in which corresponding quadrants of each field—the

upper or lower half of one temporal, and the upper or lower half of the other nasal—have been lost. These are generally caused by cortical or sub-cortical partial lesions of one occipital lobe, destruction of the part above the calcarine fissure leading to loss of the lower quadrants and *vice versâ*. Figs. 62 and 63 show the probable representation of different portions of the field in the visual cortex according to Gordon Holmes and Lister.

Homonymous defects in the visual fields are found associated with lesions of the temporal lobe owing to the fact that a ventral band of the optic radiations, the inferior longitudinal fasciculus, passes first forwards and then backwards in the temporal lobe in its course from the external geniculate body to the occipital lobe (Fig. 219, 5). Partial hemianopia, *i.e.*, more or less quadrantic defects, are then commoner than the typical homonymous hemianopia; and the defect is usually greater on the side of the lesion. Subjective sensations of smell are an important symptom in these cases, and are due to the involvement of the uncinate process of the hippocampal gyrus.

Lesions of the Optic Tract. In this case, since the afferent pupillary fibres part company with the visual fibres before the latter enter the external geniculate body (*vide* p. 60) Wernicke's hemianopic pupil reaction should be present (*vide* pp. 61, 93). It must be remembered, however, that this reaction is always difficult to elicit, and with the methods usually employed is seldom conclusive. More assistance in diagnosis is afforded by collateral symptoms. The proximity of the crus cerebri, third and other cranial nerves, leads to not infrequent involvement in the pathological process (*vide* p. 604). The association of hemianopia with contra-lateral third nerve paralysis and ipsilateral hemiplegia suggests a tract lesion. As a rule the fixation point does not escape in tract hemianopia. Partial atrophy of both optic nerves manifests itself by pallor of the discs in these cases, preceded in cases of raised intracranial pressure by papillœdema. The lesion is usually syphilitic meningitis or gumma, tubercle or tumour of the optic thalamus or temporo-sphenoidal lobe; softening and hæmorrhage are rare.

Lesions of the Optic Chiasma. Bitemporal hemianopia is usually caused by tumours in the region of the sella Turcica; these press upon the chiasma, so that the fibres going to the nasal halves of each retina are destroyed. (Fig. 219, 3). Most commonly these are tumours of the pituitary body itself; but suprasellar tumours, particularly craniopharyngiomata derived

from Rathke's pharyngeal pouch and meningiomata must be considered.

Disease of the pituitary body may manifest itself in forms which are attributed to (1) hyperpituitarism, (2) hypopituitarism, and (3) dyspituitarism. The organ consists of a glandular anterior lobe, and a posterior lobe, composed of nervous tissue, covered anteriorly by a glandular veneer, the pars intermedia. The anterior lobe appears to be specially concerned with skeletal growth, the posterior, including the pars intermedia, with tissue metabolism: *i.e.*, over-activity of the anterior lobe causes excessive growth in the bones; over-activity of the posterior lobe leads to emaciation and glycosuria with polyuria; diminished activity of the posterior lobe leads to adiposity, sugar tolerance, sexual infantilism, low pulse, low temperature, &c. (Cushing). Hyperpituitarism in infancy causes gigantism, in later life acromegaly. Hypopituitarism causes adiposity and persistence of skeletal and sexual infantilism when originating in childhood, adiposity and reversion to sexual infantilism, with development of feminine characteristics in the male, when originating in the adult (Fröhlich's syndrome). Hyperpituitarism often gradually gives place to hypopituitarism; mixed or transition cases exhibit features of both states (dyspituitarism). The adiposity associated with defective action of the posterior lobe is accompanied by excessive sugar tolerance, *i.e.*, 300 or 400 gms. of glucose or lævulose can be assimilated without the development of glycosuria, whereas about 100 gms. is the normal amount. There is reason to think that some of the symptoms, *e.g.*, somnolence, adiposity, low temperature and possibly sexual impotence, are to be attributed to disorder of the neighbouring hypothalamus rather than to the pituitary gland itself. Changes in the pituitary body are accompanied by changes in other ductless glands, such as the thyroid, pancreas, testicles and ovaries.

Enlargement of the pituitary body, whether from functional hyperplasia, adenoma, or malignant growth, leads to visual defects in about 80 per cent. of cases (Cushing), due to pressure upon the chiasma, which lies immediately above it (Figs. 220, 221), and upon the inner sides of the optic tracts. The earliest visual symptom may be a unilateral central scotoma, simulating retrobulbar neuritis (Nettleship), for one side is usually compressed before the other. This may be followed by homonymous hemianopia from pressure on one tract, or rarely by *altitudinal hemianopia*, *i.e.*, loss of the upper or more rarely lower halves of the fields, from pressure upon the chiasma; early loss in the upper half of the field may be caused by intra-

or extra-sellar tumours; early loss in the lower half is in favour of a supra-sellar tumour (Brouwer). More commonly bitemporal hemiachromatopsia, passing into a complete hemianopia, supervenes. The field does not show the accurate delimitation characteristic of homonymous hemianopia, but gradually contracts from the temporal side inwards and from

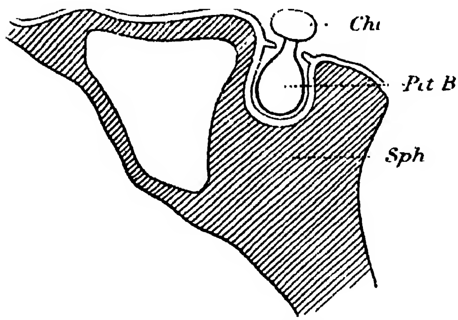


FIG. 220.—Antero-posterior section of sella Turcica. *Pit. B.*, pituitary body. *Sph.*, sphenoid. *Chi.*, chiasma.

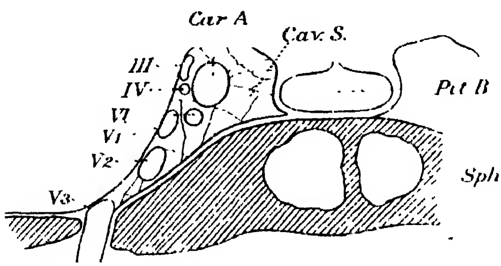
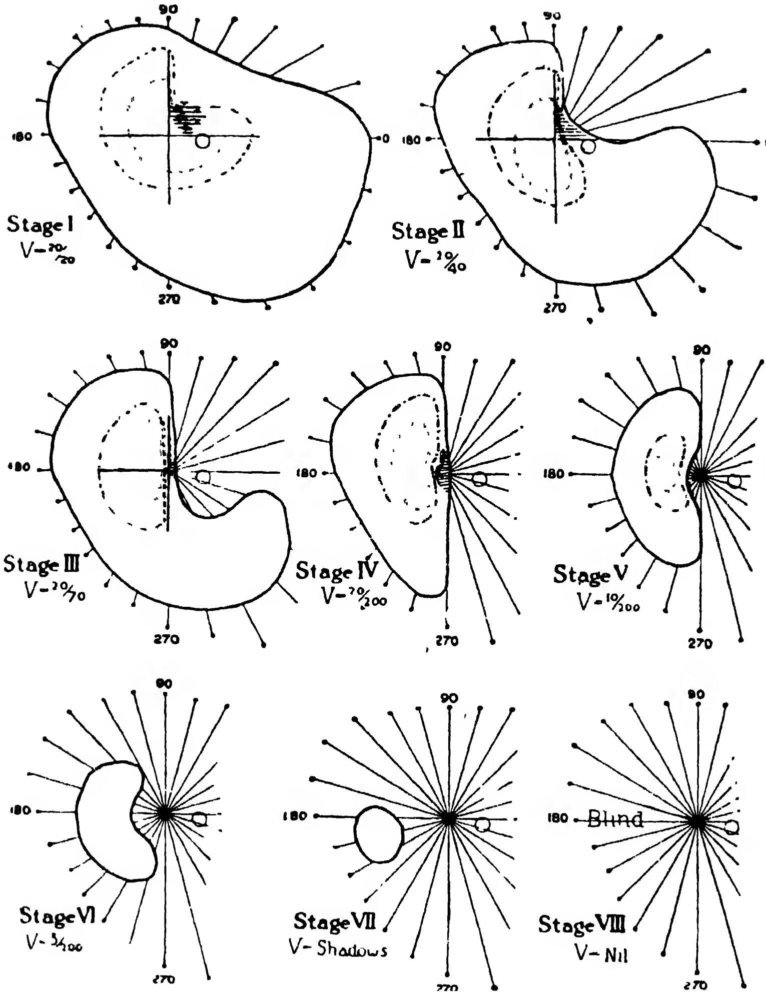


FIG. 221.—Transverse section of sella Turcica. *Pit. B.*, pituitary body. *Sph.*, sphenoid. *Cav. S.*, cavernous sinus. *Car. A.*, internal carotid artery. *III*, *IV*, *VI*, third, fourth and sixth nerves. V_1, V_2, V_3, first, second and third divisions of the fifth nerve.

above downwards, finally involving the nasal field from below upwards and leading to complete blindness in the eye affected (Plate XX). Then, or at a much earlier stage, the vision of the other eye becomes affected in a similar manner. If the second eye becomes affected before vision is lost in the first the fields show bitemporal hemianopia, but one eye is almost invariably more affected than the other, owing to the asymmetry of the growth. Complete temporal hemianopia in

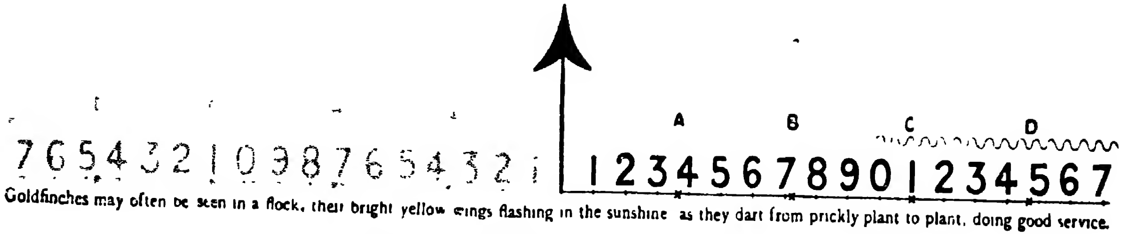
PLATE XX.



Showing the eight stages of a progressing right temporal field defect in pituitary disease. (Harvey Cushing and Clifford B. Walker.)

[*To face p.* 412.

PLATE XXI.



Maddox's scale to measure latent insufficiency or excess of convergence at the distance of 25 cm. with a prism of 12° (*i.e.*, 6° deviation or 6Δ) base upwards before the R. eye. The figure to which the lowest arrow points indicates in degrees the amount of latent convergence (black letters) or divergence (red letters) which may be present. The figures under the waved line are—teens (ten, twelve, thirteen, &c.) and the capital letters, A, B, C, D, represent metre angles for an interocular distance of 64 mm.; if the lower arrow point to black A there is 1 metre angle (1 m.a. or 1 M) of convergence, if to black B, 2 m a., &c. The printed matter under the numbers is introduced to ensure accurate accommodation for the distance (25 cm.).

one eye, for example, may be associated with temporal achromatopsia in the other. Such cases emphasise the importance of charting the colour fields in all cases. A considerable proportion of cases show homonymous hemianopia, due to pressure and traction on one optic tract. Variations in the progress of the visual defect are not uncommon. In some cases headache disappears, other symptoms are ameliorated, and the vision ceases to deteriorate. Some of these cases are probably to be attributed to cysts which have ruptured spontaneously.

If one eye is blind the history may reveal the fact that the field was lost earliest on the outer side. In cases of acromegaly, the enlargement of the jaw and characteristic facies, the large hands, the loss of sexual desire, and the presence of impotence or amenorrhœa, render the diagnosis easy. Tumours of the hypophysis are less readily diagnosed, but here also loss of sexual power is usually an early symptom, often accompanied by excessive subcutaneous fat. In all cases a skiagram of the skull should be taken ; the sella Turcica will be found often, though by no means invariably, enlarged. Associated signs are slight proptosis and paralysis of ocular nerves, generally the first division of the fifth, causing pain, and the third nerve. This is of diagnostic importance, since the sixth nerve is more commonly affected in intracranial lesions. As already mentioned, Fisher regards Leber's disease (*vide* p. 400) as probably due to pituitarism.

Some cases improve when treated with thyroid extract, others with pituitary extract, but spontaneous variations in the amount of visual defect are common. Usually the cases come under observation at a late stage when over-activity has given place to insufficiency. Consequently, treatment with active extracts of the gland or of the anterior lobe is indicated. If, as is usual, the progress of the visual defect remains unmitigated the question of operation arises, since otherwise total blindness from optic atrophy is inevitable. The transfrontal route is preferred by neuro-surgeons, and mortality has been much decreased by improved technique. Death may occur from post-operative hyperpyrexia.

A chronic *chiasmal arachnoiditis*, due sometimes to syphilis but more often of obscure origin, may also cause bitemporal hemianopia due to compression of the chiasma by fibrous cicatricial bands. The same field defect has also resulted from antero-posterior injury to the chiasma in fracture of the base of the skull.

Binasal hemianopia is very rare, if, indeed, it can be said to

occur at all in typical form. It necessitates two lesions, one on each side of the chiasma, destroying the fibres to the temporal halves of each retina while leaving the nasal fibres intact. In cases which have been reported there has usually been increased intracranial pressure with choked discs, and the condition has been attributed to distension of the third ventricle, causing the optic nerves to be pressed downwards and outwards against the internal carotids. Other cases have been referred to atheroma of the carotids or posterior communicating arteries.

Cases have been described in which there has been loss of half of one field and depression of vision progressing to blindness in the other. These are due to a lesion at the point where one optic nerve meets the chiasma (Fig. 219, 2). At this point, the crossed fibres from the opposite side loop forward into the optic nerve so that they also are involved in a lesion affecting this nerve.

Amblyopia (ἀμβλύς, blunt) and **Amaurosis** (ἀμαυρός, dark) are the terms used for partial and complete loss of sight respectively in one or both eyes. They are not used of all cases of partial or complete blindness, but have become restricted to certain forms of a more or less indefinite character devoid of ophthalmoscopic or other marked objective signs.

Unilateral amblyopia is usually either *congenital* (*vide* p. 574) or from psychical suppression of the retinal image—*amblyopia ex anopsia* (*vide* p. 574): these varieties are discussed elsewhere. Unilateral amblyopia may be due to high refractive errors in the eye. It is then not a true amblyopia, since correction with suitable glasses in early life cures the condition if sufficient perseverance is exercised. In older people glasses often fail: this may be attributed either to the development of a true amblyopia from disuse or, more probably, to defective receptivity of the higher centres. Unilateral amblyopia is also a symptom of retrobulbar neuritis (*q.v.*).

Bilateral amblyopia is found in the various forms of toxic amblyopia (*q.v.*). Bilateral amaurosis occurs in uræmia and in meningitis. Both amblyopia and amaurosis occur in hysteria.

Uræmic Amaurosis occurs particularly in acute nephritis, *e.g.*, in pregnancy, after scarlet fever, &c., but is also found with chronic nephritis. The onset of blindness is sudden or rapid (8–24 hours); it is bilateral and complete. The fundi show no changes, unless, as in some cases, there is a coincident albuminuric retinitis. Vision usually improves in 10–18 hours, and is fully restored in about 48 hours, especially if a lumbar puncture is done. In cases during pregnancy there is usually

eclampsia. In uræmic amaurosis the pupils are dilated, but usually react to light. It is probably due to circulation of toxic material which acts upon the cells of the visual centres. The retained reaction of the pupils to light shows that the lower centres are relatively immune. Exophthalmos sometimes occurs in cases of nephritis in which uræmia is present or imminent, and may therefore be of some prognostic significance. It is accompanied by pain and limitation of movement of the eyes, and is probably due to œdema of the orbital tissues.

Hysterical amblyopia, as might be expected, exhibits protean manifestations. It may be unilateral, but is more commonly bilateral. There is usually concentric contraction of the fields, with or without colour defects. A spiral field is very characteristic, *i.e.*, the field continually diminishes while it is being taken, so that it may be finally limited to the fixation point. The patients, however, get about perfectly well unaided, an impossibility in cases of genuine highly contracted fields (*vide* p. 375). The condition is sometimes called anæsthesia of the retina, an undesirable designation. Sometimes there are irritative symptoms—blepharospasm, blinking, lacrymation, &c.—hyperæsthesia of the retina. The pupillary reactions are perfect, affording an invaluable objective diagnostic sign. The prognosis in hysterical amblyopia is good, though treatment is usually tedious. The chief difficulty consists in eliminating organic disease, such as retrobulbar neuritis, injury, embolism of the central artery of the retina, sympathetic ophthalmia, and so on.

Amaurosis fugax is a term given to sudden temporary failure of sight, and is due to various causes. In its simplest form it occurs in normal people on rising suddenly from the sitting or recumbent to the upright posture. It is then due to the effects of gravity and is merely momentary, accompanied by slight giddiness and even faintness. In dangerous form it occurs in aeroplane "black-out." Transient blindness, seldom complete, occurs as a prodromal symptom of obstruction of the central artery of the retina, and is probably due to spasm of the arteries or to the effects of changes in blood pressure associated with arteriosclerosis. It has been met with in people with mild signs of Raynaud's disease. Temporary amblyopia also occurs in migraine (*q.v.*), and in early stages of papillœdema (*q.v.*) from increased intracranial pressure.

Scintillating Scotomata of various kinds occur in migraine. In typical migraine the patient feels unusually well before the attack. A positive scotoma appears in the field of vision;

while obscuring sight it has a peculiar shimmering character. It gradually increases in size until predominantly one-half of the field is clouded, the fixation point remaining relatively clear. In the dark field there are often seen bright spots and rays of various colours, and these are often arranged in zig-zags and are then called fortification spectra (*teichopsia*). Both half fields are often affected, so that there is homonymous hemianopia. In other cases the whole field becomes clouded, but usually even so the fixation point is seen momentarily, and then becomes obscured until the eyes are moved to a fresh spot. Vision usually clears in about a quarter of an hour. The attack is soon followed by violent headache, generally limited to the opposite side of the head to the hemianopic field (*hemicrania*), and accompanied by nausea and even sickness ("bilious attack"). During the attack there is frequently numbness in the mouth and tongue and slight aphasia. Attacks occur periodically, but vary greatly in number and severity. In mild attacks the scotoma or slight aphasia may occur without the headache and *vice versâ*. In older persons subject to migraine only parts of the typical attack may occur, *e.g.*, the scotoma with little headache, or a migrainous headache without scotoma.

Migraine is to be attributed to vaso-motor changes in the brain. Vaso-dilatation, associated with a feeling of well-being, is followed by vaso-constriction, especially in the occipital lobes. There is often a copious secretion of urine of low specific gravity during the attack. Constriction of the retinal arteries has been described during the scotomatous stage, but it is very doubtful if it ever occurs.

Migraine occurs chiefly in highly-strung people and may sometimes be accentuated, if not caused, by chronic forms of peripheral nerve irritation, especially such as are due to astigmatism, anisometropia, &c. Some cases, but by no means all, have been cured by accurate correction of the errors of refraction and the wearing of suitable glasses. A sedentary life, with constipation and insufficient exercise, conduces to the attacks. Rest, warmth, and sleep are the best cures for the actual attacks. They can sometimes be warded off or alleviated by aspirin or ergotamic tartrate tablets. Nitroglycerin or amyl nitrite have been used, but are not reliable cures.

Occasionally people who suffer from ordinary migraine have attacks in which, without any scotoma, the headache is followed by partial paralysis of the third nerve (*ophthalmoplegic migraine*)

on the same side as the hemicrania. Slight ptosis, diplopia, and sluggishness of the pupil reactions continue for some hours and gradually disappear. The paresis is worse and persists longer with succeeding attacks, and has sometimes eventually become permanent. Probably most of these cases are not migrainous, but due to some organic nerve lesion, *e.g.*, pressure on the nerve by a congenital aneurysm of the circle of Willis, and some of the patients have died from subarachnoid hæmorrhage (*vide* p. 609).

Night-blindness occurs *par excellence* in retinitis pigmentosa (*q.v.*) and in xerophthalmia (*q.v.*). In rare cases it is a familial congenital affection. It is also found in endemic form, especially after exposure to bright sunlight in hot countries, *e.g.*, amongst soldiers and sailors. The patients are usually overwrought, as by long marching, or debilitated, as by scurvy, fasting in Lent, &c. The condition generally improves rapidly if the eyes are protected and the nutrition attended to. The affection is purely local, as is shown by the fact that covering one eye with a bandage during the day has been found to restore sight enough for the ensuing night's watch on board ship, the unprotected eye remaining as bad as ever. Night-blindness is to be attributed to interference with the functions of the retinal rods, due to deficiency in visual purple. In xerophthalmia and the endemic cases the symptom is a manifestation of deficiency of fat-soluble vitamin A in the diet, and therefore cod-liver oil is specially indicated. It also occurs in diseases of the liver, especially cirrhosis. Soldiers often complain of it, but not infrequently it is a functional nervous disorder in these cases, associated with other symptoms of neurosis or malingering.

Day-blindness occurs in some cases of congenital amblyopia. It also occurs in all cases of reflex blepharospasm. In less noticeable form it occurs in lesions affecting the conducting paths of visual impulses, such as tobacco amblyopia, retrobulbar neuritis, and the early stages of optic atrophy. Patients suffering from these disorders often see relatively, and sometimes absolutely, better in a dull than in a bright light.

Coloured Vision is sometimes complained of, and red is the colour usually noticed. *Erythropsia* occurs particularly after cataract extraction if the eyes are exposed to bright light and are over-strained. In these cases it may persist for several hours or days. Objects look red, but the visual acuity is not affected, and no permanent damage results. Patients should be warned of the possibility of erythropsia, as it is somewhat alarming and suggestive of hæmorrhage. It is met with also

in snow blindness. Red vision is sometimes complained of by neurotic hypermetropic children. Chromatopsia also occurs in some cases during the resolution of optic neuritis when the ensuing atrophy is not complete. In normal people black print will sometimes suddenly turn deep red: this is due to strong lateral light entering the eye through the sclerotic.

Metamorphopsia; **Micropsia**; **Macropsia** or **Megalopsia**; **Photopsiæ** (*vide* pp. 339, 349).

Muscæ volitantes (*vide* p. 330).

Colour Blindness or **Achromatopsia** may be congenital or acquired. *Acquired colour blindness*, partial as in cases with relative scotomata, or complete as in disease of the optic nerve, has been referred to incidentally in treating of the various disorders of the eye in which it occurs. It may also be a symptom of disease of the central nervous system, especially perhaps the lowest parts of the occipital cortex. In most diseases of the retina and choroid, *e.g.*, detached retina, changes in colour perception affect mostly the blue end of the spectrum. Slight diminution in acuity of perception of these rays is caused normally, owing to their physical absorption, by the increase of amber pigment in the nucleus of the lens (*blue blindness*), and this may be abnormally great in sclerosing lenses (black cataract). It has been said to affect the pictures of artists in their old age (Liebreich). Slight absorption of rays of short wave-length is normal at the macula, owing to the yellow pigment present here; it varies in different individuals.

Congenital Colour Blindness occurs in two chief forms, total and partial. The former is very rare and is generally associated with nystagmus and a central scotoma. All colours appear grey, of different brightness. The spectrum appears as a grey band exactly like the normal scotopic spectrum (*vide* p. 68), and like it with the maximum brightness at 510 $\mu\mu$. It does not change, except in increase of brightness, when the intensity is increased, but at moderately high intensity photophobia occurs. It is probable that total colour blindness is caused by defective development of cones or their complete absence.

The partial form is seldom discovered unless specially tested for, since the subjects compensate for their defect by attention to shade and texture, combined with experience. Gross cases occur in 3 to 4 per cent. of males, but are rare in females (0·4 per cent.); slighter cases are quite common in males. It is an inherited condition, being transmitted through the female, who is usually unaffected. In most cases reds and greens are confused, so that the defect causes grave

danger in certain occupations, *e.g.*, railway signalmen, engine-drivers, and sailors. The red-green cases fall into two chief groups, protanopes and deuteranopes. For the former the red end of the spectrum is much less bright than for normal people and is often actually shortened. These groups are explained on the Young-Helmholtz theory by the hypothesis that one of the primary sensations (*vide* p. 69) is lacking. They are therefore often said to have dichromatic vision. In the protanopes the so-called red sensation is said to be absent, and they are called red-blind; in the deuteranopes the green sensation is absent and they are called green-blind. On Hering's theory both groups are varieties of red-green blindness, which is explained as due to absence of the red-green substance (*vide* p. 69). In both groups the defects may not be complete and these cases are called protanomalous and deuteranomalous respectively. Since, on the Young-Helmholtz theory, all three sensations are represented, though one is defective, these are said to have anomalous trichromatic vision. It is clear that theoretically there might be other cases of colour blindness due to absence of the blue sensation or the blue-yellow substance, and such have been described, but are very rare (tritanopes).

There are two objects to be aimed at in testing for colour blindness: (1) the exact scientific nature of the defect; (2) whether the subject is likely to be a source of danger to the community. The first is exhaustive and may be the only means of arriving at the second, especially in the anomalous trichromats. In the more difficult cases, besides the simpler tests, more stringent tests with a pure spectrum must be employed. In them only those of large experience will gather any useful information from the names given by the subject to various colours, for these are named chiefly by reference to their various brightnesses and the answers appear to be quite inconsistent. In testing for danger only, it is obvious that the names given to the colours are of value, for if a man repeatedly calls red green or *vice versâ* he is clearly unsuited to be an engine-driver or look-out man on a ship.

Whatever the object in view, several tests should be employed. For the spectrum tests the student must be referred to special monographs on colour vision. The following are the chief other tests.

(1) *The Lantern Test.* The subject names various colours shown by a lantern, and is judged by the mistakes he makes.

Much here depends upon the size of the apertures of the lantern (*i.e.*, the size of the retinal areas stimulated) and the nature and intensity of the light source. Many lanterns are worse than useless. The best is that used by the Board of Trade, which has now been adapted to electric light. Edridge-Green's lantern is efficient if used by an expert.

(2) *Holmgren's Wools.* These consist of a large selection of skeins of coloured wools, and the candidate is required to make a series of colour matches.

Test I. consists in presenting to the candidate a pale green sample and telling him to select from the heap of wools all those which seem to correspond in colour. If he is colour blind he will probably select several of the "confusion colours"—greys, buffs, straw colour, &c.—as well as greens. He is next given a rose colour (II.): if he matches this with blues or violets he is red-blind; if with greys or greens he is green-blind. He is then given a bright red skein (III.): if he is red-blind he will choose dark greens and browns, if green-blind pale greens and browns. Sir William Abney has recommended the addition of two other tests. IV. is a purple skein: if the candidate is colour blind he will probably select any shade of blue or green, also pinks and greys. V. is a yellow skein: the colour-blind candidate will probably select greenish-yellows, light yellow-greens, fawns and pinks. In blue-blindness purples, red and orange are confused in test II.

Holmgren's wools have been much criticised, but if the tests are properly carried out gross defects of colour vision are easily recognised and an expert will be put on his guard in almost every case of even minor defect.

(3) *Stilling's Tests.* These consist of coloured lithographic plates, in which bold numerals are represented in dots of various tints set amid dots of the same size but of tints which are most readily confused with those of the figures by colour-blind people. Normal trichromats can easily read the numbers, some of which are indistinguishable to the various types of colour blind. Ishihara's tests are a modification and, in some respects, an improvement on Stilling's: they include tests in which the numbers can be read by the colour blind, but not by the normal sighted.

(4) *Nagel's Anomaloscope.* This is an instrument in which on looking down a telescope a bright disc is seen, divided into two halves by a horizontal line. One half is illuminated by

light of the sodium line of the spectrum (yellow), and this has to be matched by a mixture of red (lithium line) and green (thallium line) in the other half. By turning a screw the relative amounts of red and green in the mixture can be varied. By turning another screw the brightness can be varied.

Defects of colour vision have led to much acrimonious discussion. Their detection may be easy, but is often difficult. No theory which has yet been brought forward is wholly satisfactory, and no single test is infallible.

Malingering. Cases occasionally occur of men who hope to gain some advantage by pretending to be blind. It is rare for complete blindness to be assumed, and such cases can only be detected by constant watching of the person's behaviour. When one eye is said to be blind, in spite of absence of sufficient objective evidence to account for the condition, the demonstration of malingering resolves itself into a contest of wits between the surgeon and the individual. Many tests have been devised, and several should be employed in each case.

(1) A low concave or convex glass (O. 25 D) is placed before the "blind" eye, and a high convex (+ 10 D) before the "good" eye, and the examinee is told to read the distant types. If he succeeds malingering is proved.

(2) A prism is placed base downwards before the "good" eye and the examinee is told to look at a candle. If he admits to seeing two candles malingering is proved.

(3) The surgeon stands behind the patient and covers the "blind" eye with his hand, at the same time holding a prism of 10 degrees base down before the "good" eye in such a manner that the edge of the prism passes horizontally across the centre of the pupil. Uniocular diplopia results. The surgeon then simultaneously removes his hand from the "blind" eye and shifts the prism upwards so that the whole pupil is covered by it. If the examinee still admits to seeing two candles malingering is proved.

(4) While the examinee looks at a candle a prism of 10 degrees is placed base outwards before the "blind" eye. If the eye moves inwards in order to eliminate diplopia it is not blind.

(5) Snellen's coloured types may be employed. The letters are printed in green and red. If a red glass is placed before the "good" eye, and the patient reads all the letters, the other eye is not blind, for the eye looking through the red glass can only see the red letters. Care must be taken in this test that

the red glass cuts off all the rays from the green letters as tested by the surgeon's own vision.

Word-blindness occurs as a not very uncommon congenital defect—0·1 per cent. of elementary school children (Thomas). It is much commoner in boys than girls. Owing to backwardness in learning to read the children are often brought to the ophthalmic surgeon, visual defect being suspected. In spite of normal fundi and often quite normal acuity of vision, the patients fail to recognise printed or written words. The auditory memory of words is unimpaired, and generally numerals and music can be read. Hence the patients learn well orally, and are good at arithmetic. They are often quite intelligent and may be wrongly punished for inattention and stupidity. The condition sometimes runs in families. The defect is not necessarily complete, and much improvement can be obtained by careful individual tuition and perseverance.

CHAPTER XX

Intra-Ocular Tumours

INTRA-OCULAR tumours are rare, but of great importance, since they are usually malignant and endanger the life of the patient.

Tumours of the Uveal Tract. The malignant tumours of the uveal tract have long been considered mesodermal and have become classically known as sarcomata. The majority are pigmented, but the pigment is not essential to their nature. Evidence has recently been brought forward to show that simple nævi and malignant melanomata of the skin are neuro-ectodermal in nature, being derived from the cells of the sheaths of Schwann (Masson), and it has been contended that the common uveal tumours have a similar origin (Theobald): for this reason these tumours are now frequently referred to as "malignant melanomata."

Tumours of the Iris. It is not uncommon to see irides with dark brown spots (melanomata), due to congenital aggregations of retinal pigment epithelium. As a rule these are benign, but occasionally they take on malignant proliferation. Any increase of size must be watched with suspicion. Pigmented nævi also occur rarely.

Sarcoma, composed of pigmented or unpigmented spindle-shaped or round cells, occurs as an isolated nodule, but is very rare. It grows rapidly, and if left attacks the corneo-sclera and perforates the globe. It gives rise to metastases from which the patient dies. Although it is the only new growth of importance met with in the iris, it may be diagnosed from gumma or tubercle only with difficulty. The chief points of difference are the absence of inflammation as shown by synechiæ, &c., the absence of satellites, the frequency of pigmentation, and the rapid increase in size.

Treatment. The growth should be watched for a short time, and if found to increase in size should be removed by iridectomy if this is feasible. The specimen is subjected to expert microscopic investigation. If the growth is malignant and has not been completely removed or shows signs of recurrence it is safest to exercise the eye at once, though successful treatment

by radiation has been recorded. If completely removed the prognosis is good.

Sarcoma of the Choroid is not so rare as sarcoma of the iris. The growth arises from malignant proliferation of the stroma cells of the outer layers. It forms at first a lens-shaped mass, raising the retina over it. By the process of growth increased tension is thrown upon the elastic membrane of Bruch, which finally ruptures. The cells then proliferate through the opening and form a globular " head " in the subretinal space, separated from the mass in the choroid by a narrow " neck " (Fig. 222).

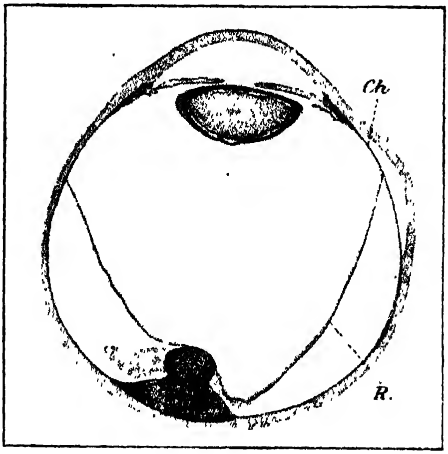


FIG. 222.—Section of sarcoma of the choroid ($\times$ 9), showing the typical mushroom shape. *Ch.*, pars plana of ciliary body, continuous posteriorly with the choroid, behind *R*, retina.

The retina remains in contact with the tumour at the summit of the head, but is detached from the choroid at the sides, the intervening space being filled with albuminous fluid. The growth may be in any situation, and the fluid may sink down to the lowest part of the eye, forming there a " simple " detachment isolated from that over the tumour. As the tumour progresses the retina is more and more detached, until no part remains *in situ*. The nutrition of the lens then suffers, so that it becomes opaque. The tumour may fill the globe before perforating the sclerotic, or this may occur relatively early along the perivascular spaces of the vortex veins or ciliary vessels. The orbital tissues then become infiltrated. The

lymphatic glands are not affected, but metastases occur in the liver and elsewhere.

The growth is usually pigmented—melanotic sarcoma; rarely unpigmented—leucosarcoma. The distinction is relatively unimportant pathologically. The pigment is chiefly melanin, but hæmatogenous pigmentation occurs after hæmorrhages. Metastases from melanotic growths are often unpigmented. The cells are usually spindle-shaped. They may be cylindrical or palisade-like, arranged in columns, or around blood vessels (angiosarcoma), or the cells may be endothelial. Most sarcomata are mixed-celled. Silver staining reveals a variable amount of argyrophile "reticulin" fibres, generally most in spindle-celled sarcomata. There is evidence that those with most reticulin are least malignant.

The course of sarcoma of the choroid is commonly divided into four stages: (1) the quiescent stage; (2) the glaucomatous stage; (3) the stage of extra-ocular extension; (4) the stage of metastasis. This is probably the typical chronological order of events, but secondary glaucoma may arise at a very early stage or be delayed until after extra-ocular extension has taken place, and there is evidence to show that metastases may occur at an early stage.

The cause of the glaucoma is obscure: in most cases it is due to the lens and iris being forced forwards, so that the angle of the anterior chamber becomes blocked. In other cases, particularly those of early onset, obstruction to the venous outflow from the eye is the probable explanation, the tumour being in some instances so situated as to press upon a vortex vein.

Sarcoma of the choroid usually occurs in adults between forty and sixty. It is always primary, single, and unilateral. The patient may come under observation in the early stage when there is only detachment of the retina. The earliest cases to seek advice are those in which the tumour is near the macula, since vision is then most strikingly affected. In other cases the tumour has usually attained a considerable size, and the patient may apply for treatment for relief of the pain of glaucoma.

It is of the utmost importance that the cause of the detachment of the retina should be diagnosed in these cases. We have already seen that "simple" detachment of the retina is most frequently found in myopic eyes or after a blow, though many cases are apparently "idiopathic." In simple detachment the lower part of the retina is usually affected, though not always. There is therefore presumptive evidence—of only

slight weight—that a detachment in the upper part is due to a tumour of the choroid. In detachments of considerable size the tension of the eye is normal or subnormal in the absence of a growth ; if the tension is raised quite definitely a growth may be diagnosed almost with certainty. A simple detachment shows numerous, more or less parallel, folds, and undulations can be seen to travel over the surface when the eye moves. The detachment at the summit of a tumour is usually rounded and fixed, though in the surrounding parts it may show all the signs of a "simple" detachment. Patches of pigment upon the rounded part support the diagnosis of a tumour, but pigment disturbance, more particularly at the periphery, is not uncommon in simple detachment. Rarely a system of blood vessels having an entirely different mode of distribution from the retinal vessels can be made out between the latter vessels. This is the most positive evidence of growth, but it is only occasionally seen. A very small, round detachment in the macular region or upper part of the globe is almost certain to be due to a tumour of the choroid. If the detachment is sufficiently anterior, transillumination with a specially devised lamp will afford assistance in diagnosis. After anæsthetising the eye and dilating the pupil with homatropine the minute lamp is placed in contact with the eyeball as nearly as possible over the situation of the growth and the pupil observed in the dark room. In cases of doubt when the suspected neoplasm is situated behind the equator it is justifiable to incise the conjunctiva and Tenon's capsule and pass posteriorly over the sclera a specially constructed small transillumination lamp to the site of the growth. A simple detachment is transparent, a choroidal growth opaque.

Diagnosis may be extremely difficult if the patient is first seen when glaucoma has already supervened. Dependence must then be placed largely upon the history. Defective vision may have been noticed, but the premonitory haloes of glaucoma have been absent, and vision has gone from bad to worse without remissions. One eye only is involved. The other may be perfectly normal, or at least not of the glaucomatous type with small cornea and so on, and the field of vision in this eye will show no contraction on the nasal side. The affected eye will probably have no perception of light, so that if any doubt remains it should be excised.

In the diagnosis two rare simple tumours must be kept in mind, particularly in the early stages. A choroidal nævus appears as a bluish patch with somewhat feathered edges, usually about the

size of the optic disc and situated near the posterior pole of the eye. It is congenital and symptomless but, like nævi elsewhere, may occasionally assume malignant characteristics. A cavernous hæmangioma of the choroid, another rare tumour of congenital origin and of exceedingly slow growth, is also usually situated near the disc. It has a greyish hue and steep margins and often causes a retinal detachment. It is frequently associated with dilated vessels in the fundus or hæmangiomata elsewhere.

Treatment. The eye should be excised as soon as possible after arriving at the diagnosis of sarcoma. Although such growths rarely travel down the nerve, it is wise to cut it as long as may be. If the growth has already burst through the globe the orbit should be exenterated, or irradiated with X-rays or radium. When the affected eye is the only seeing eye, the choice of excision or treatment by diathermy or suturing radon seeds to the sclera over the site of the neoplasm (*vide* p. 431) should be put before the patient.

The disease is invariably fatal, usually within five years, if not eradicated by operation: metastases may be delayed for ten years or more. Prognosis is fair if the tumour is small and entirely intraocular, especially if it contains much reticulin (*vide* p. 425).

Flat Sarcoma of the Choroid. In rare cases the choroid becomes infiltrated with sarcoma cells which cause a uniform thickening and shallow " detachment " of the retina. These are probably endotheliomatous, spreading along the lymphatic spaces of the choroid in the same manner as secondary carcinoma (*q.v.*).

Sarcoma of the Ciliary Body is fundamentally of the same nature and gives rise to the same symptoms as sarcoma of the choroid, the differences being only those dependent upon the anatomical disposition of the parts. Thus the retina, being here more adherent to the underlying uvea, and being reduced to a double layer of epithelial cells, is not detached. When the growth has spread to the choroid the retina proper becomes detached. The tumour may attain considerable size before it causes symptoms, which are then referable to displacement or distortion of the lens and interference with the ciliary muscle. The ciliary circulation is impeded, and conspicuous dilatation of one or two anterior perforating ciliary vessels should always arouse suspicion. The growth may invade the angle of the anterior chamber. It then has the appearance of an iridodialysis, a dark crescent showing at the root of the iris. That it is not an iridodialysis is shown by the fact that no reflex can

be obtained through it on illuminating with the ophthalmoscopic mirror and from the absence of history of a blow. In the case of a leucosarcoma the crescent may be yellowish, but vessels will usually be visible upon the surface, and these render the diagnosis easy. The growth may be visible by oblique illumination with a widely dilated pupil, and is opaque to transillumination.

Sarcoma of the ciliary body is less common than that of the choroid. The treatment and prognosis are the same.

Ring or Annular Sarcoma of the Ciliary Body resembles flat sarcoma of the choroid in its infiltrating character. It is very rare.

Epithelial hyperplasia of the ciliary processes is not uncommon in old eyes. Rarely malignant epitheliomata occur; also growths resembling embryonic retina in growing people (diktyoma). They cause the same clinical signs as sarcoma.

Secondary Carcinoma of the Choroid occurs sometimes in late stages of scirrhus of the breast, more rarely in cancer of other organs. There is obscuration of vision, and ophthalmoscopic examination reveals a widespread shallow detachment of the retina, usually at the posterior pole. The disease is nearly always bilateral, and as it is usually only one of many metastatic deposits and the patient is generally in the last stages of general carcinomatosis, no special treatment is indicated.

Retinoblastoma (*Syns.—Glioma retinæ, Neuroepithelioma retinæ*) has hitherto been more commonly known as glioma retinæ. True gliomata, however, *i.e.*, malignant proliferations of neuroglia such as occur in the brain and optic nerve, are very rare in the retina. The usual malignant growth of the retina is due to proliferation of neural cells, and is better termed retinoblastoma.

It is confined to infants, and is probably always congenital, though it may remain quiescent or pass unnoticed until the fifth or sixth year or even later. The disease is rare; the second eye is affected, independently and not by metastasis or continuity *viâ* the chiasma, in about one-fourth of the cases, but frequently the growth cannot be recognised even on careful examination until after months or even years. Several children of the same family are sometimes affected.

The child is brought to the surgeon on account of a peculiar yellow reflex from the pupil, sometimes called "amaurotic cat's eye." If left untreated retinoblastoma runs through the same stages as sarcoma of the choroid, viz., (1) the quiescent stage; (2) the glaucomatous stage; (3) the stage of extra-ocular

extension; (4) the stage of metastasis. The second stage results in enlargement of the globe, with apparent or real exophthalmos. Pain is severe during this stage, but is relieved when the tumour bursts through the sclerotic. Perforation often occurs at the limbus, and is followed by rapidly fungating growth. Metastasis first occurs in the preauricular and neighbouring glands, later in the cranial and other bones. Direct extension by continuity to the optic nerve (which is early affected) and brain is commoner, and metastases in other

FIG. 223.—Section of retinoblastoma (× 3). Note the rings of deeply stained cells surrounding blood vessels; also the infiltration of the anterior part of the optic nerve.

organs, usually the liver, are relatively rare (*cf.* Sarcoma of the Choroid). In most cases the first stage lasts from six months to a year.

The growth consists chiefly of small round cells with large nuclei resembling the cells of the nuclear layers of the retina; masses of these stain badly, showing that they are undergoing necrosis (Fig. 223). Among them may be found rosette-like formations of cells resembling the rods and cones. Such rosettes are also found in microphthalmia, and have been produced in embryonic eyes by irradiation and by trephining

undeveloped rats' eyes (Tansley). The growth probably originates in an island of undifferentiated embryonic retina which has failed to develop normally.

Retinoblastoma is invariably multiple (*cf* Sarcoma of the Choroid). When seen very early, as for example in the second eye, a larger mass is seen surrounded by numerous punctate satellites. Microscopically, minute deposits are seen scattered in various situations throughout the globe. It may grow principally outwards, separating the retina from the choroid ("*glioma exophytum*"), or inwards towards the vitreous ("*glioma endophytum*"). There is no fundamental distinction, but the ophthalmoscopic appearances differ in the two types. In the former the condition resembles a mere detachment of the retina ; in the latter polypoid masses may be seen stretching into the vitreous. Hæmorrhages upon the surface are not uncommon, especially in glioma endophytum.

Several conditions occurring in children may give rise to similar signs, and cause great difficulty in diagnosis. These have been grouped together under the term "*pseudoglioma.*" The chief are (1) inflammatory deposits in the vitreous, with or without detachment of the retina ; (2) tubercle of the choroid, especially the confluent type ; (3) congenital defects, due to persistence of part of the fibro-vascular sheath at the back of the lens. The first group are due to a quiet form of cyclitis, which may have passed unnoticed or may have caused slight redness of the eyeball with inflammatory deposits in the anterior chamber ; these, however, are usually soon absorbed. A history of fits, unconsciousness, attacks of screaming, ear disease, meningitis (especially post-basic), one of the acute specific fevers, syphilis, &c., may be obtained. Iritis, or the results of iritis or iridocyclitis, *e.g.*, posterior synechiæ, retraction of the base of the iris, and so on, are often present.

In all cases atropine should be instilled and both eyes should be thoroughly examined opthalmoscopically, under general anæsthesia if necessary. The tension may then be satisfactorily tested, and may afford useful information which cannot be obtained without an anæsthetic. Raised tension is in favour of retinoblastoma, lowered of pseudoglioma. Even when every precaution is taken there is a considerable group of cases in which it is quite impossible to be certain of the diagnosis. Considering that the life of the patient is at stake and that the eye is in any case useless as an organ of sight, these cases should be treated as malignant.

Treatment. The treatment is excision of the eye at the earliest possible moment. The optic nerve should be cut long, and the cut end invariably submitted to microscopical examination. If there is any doubt of extension of the disease to the conjunctiva or orbital tissues exenteration of the orbit is imperative. In cases where the diagnosis is doubtful the eye should be removed, for in inflammatory pseudoglioma the eye is destined to shrink and become unsightly. In no case should both eyes be removed at the same operation, but if one is proved by microscopical examination to contain retinoblastoma and the other contains similar nodules, it is justifiable to treat these by destruction by diathermy or radon.

In treatment by radon a 2 millicurie seed is stitched to the sclera over the site of the nodule. When the growth is situated near the macula and optic disc the radon seed or seeds may be embedded in a strip of Stent wax moulded over the appropriate site. The Stent is kept in position by stitching it to the sclera at about the equator. There is histological evidence that a 2 millicurie radon seed destroys the growth for a radius of at least 3·5 mm. around it. The number of seeds required will depend upon the size of the neoplasm; four is the maximum yet used with good results. Late sequelæ of irradiation are thin greyish exudates at the macula eighteen months, and posterior cortical lens opacities from 9 months to 8 years after treatment. Some patients are still alive eleven and twelve years after radon treatment. Radon seeds should be used only in bilateral retinoblastoma, or when excision of the eye is absolutely refused.

The prognosis of retinoblastoma, if untreated, is absolutely bad, the patient invariably dying. The prognosis is fair if the eye is removed before extra-ocular extension has occurred. In the absence of disease of the second eye the patient may be regarded as out of danger if there is no recurrence in the orbit within three years, but the remaining eye should be carefully examined under atropine at frequent intervals for a much longer period. There are several cases on record of cure after removal of both eyes.

CHAPTER XXI

Injuries to the Eye, Panophthalmitis, and Sympathetic Ophthalmia

The eye is protected from direct injury by the lids and the projecting margins of the orbit. Nevertheless, it is not exempt from foreign bodies, the action of caustics, contusions by blunt and wounds by sharp instruments.

Foreign Bodies, Burns, &c.

Foreign Bodies, which are usually small—particles of coal dust, emery, steel, &c.—may pitch upon the conjunctiva or upon the cornea. In the former case they cause sudden discomfort and reflex blinking. The foreign body sticks to the palpebral conjunctiva and is liable to be dragged across the cornea, which it excoriates. It may get floated by tears towards the inner canthus, and so into the nasal duct. Very frequently it becomes lodged at about the middle of the upper sulcus subtarsalis (*vide* p. 617), where it is most likely to irritate the cornea, or in the upper fornix. It may occasionally become imbedded in the bulbar conjunctiva. Quite large foreign bodies, such as a grain of corn, may be retained for a long time in the upper fornix and give rise to much irritation and some discharge. They are liable to be overlooked unless the upper lid is everted. They are generally imbedded in a mass of granulation tissue, which may simulate the cockscomb type of tubercle (*vide* p. 183). The wing cases of insects and the husks of seeds may adhere by their concave surfaces to the cornea, usually at the limbus, for several days or even weeks.

Fragments of aniline pencil in the eye cause much irritation and a very unsightly staining. The eye should be irrigated with a weak solution of alcohol, and glycerine drops used (Swanzy and Werner), since these substances are solvents of aniline violet (*vide* p. 185).

Particles of steel and emery are very liable to fly straight on to the cornea and penetrate into the epithelium or substantia propria. Larger particles of steel, or less commonly stone, glass, &c., may perforate the globe (*vide* p. 452). When

situated in the cornea they cause great pain and irritation. The pupil is often constricted. If allowed to remain they expose the cornea to the dangers of infection by organisms in the conjunctival sac and ulceration. This may lead to a small superficial slough being cast off, carrying the foreign body with it. The small ulcer thus formed may heal, but if virulent organisms are present, a spreading ulcer, with or without hypopyon, may develop.

It is not always easy to discover a foreign body upon the cornea. If situated eccentrically on the cornea a leash of conjunctival blood vessels will be dilated on this side and will point in the direction of the foreign body. In case of doubt the eye should be anæsthetised and the cornea thoroughly examined under oblique illumination with a loupe. The use of fluorescein will sometimes, but not always, reveal the position. In some cases the foreign body can be detected by reflecting light into the eye with a plane mirror, especially if a convex lens is used to magnify the object (*vide* p. 236).

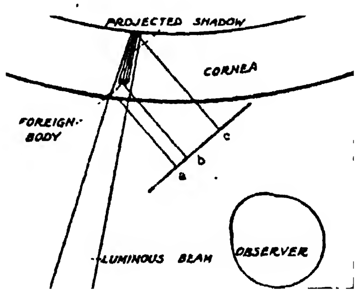


FIG. 224.—Slit-lamp illumination. (Koby.)

The binocular corneal microscope is of great assistance in determining the position and nature of the foreign body. When combined with illumination by the slit-lamp the depth of an imbedded foreign body can be measured by the aid of a micrometer, or estimated by the length of the shadow which it casts (Fig. 224).

Treatment. Foreign bodies must be removed as soon as possible, and as far as possible with antiseptic precautions. If situated in the lower fornix they are easily removed with a clean handkerchief after everting the lower lid. If not found in this position the upper lid should be everted (*vide* p. 80); the particle will generally be found in its favourite situation, and can be removed in the same manner or by passing the finger over the surface. If it is still not seen the upper fornix should be brought into view (*vide* p. 81) and the particle removed. In case of difficulty, previous application of pantocain will materially assist.

If the foreign body is imbedded in the bulbar conjunctiva it should be picked out by a needle after application of pantocain.

If a discission needle is not at hand a darning needle may be used; it should be passed through a flame first, so as to sterilise it. This little operation is performed in the same manner as removal of foreign bodies from the cornea (*vide infra*). It may be necessary to snip off the small piece of conjunctiva containing the foreign body with scissors.

Removal of foreign bodies from the cornea is effected as follows: The eye is anæsthetised and the patient seated in a chair. The surgeon stands behind the patient and holds the lids apart with the first and second fingers of his left hand, pressing slightly backwards so as to steady the globe. An assistant focusses the light upon the cornea, the patient being told to look in the direction which affords the best view of the particle. An attempt may first be made to remove the foreign body by touching it with a slip of clean blotting-paper, which exercises a capillary attraction. If this fails a sterilised spud (Fig. 225) is used. Only if this too fails after repeated efforts should a needle be resorted to. It may be a discission needle, or in default of that a darning needle (*vide supra*). The greatest care should be exercised not to scrape up the epithelium more than is absolutely necessary. Emery and steel particles cause a little ring of brown stain around them, which should be scraped off; if this is not done the patient is likely to return, under the impression that the foreign body has not been removed. If there is any sign of ulceration, *e.g.*, greyish infiltration around the abrasion, and *the patient is young, with normal tension*, a drop of 1 per cent. atropine should be instilled, warning being given that the sight will be misty for a few days. If the patient is over forty or has any signs indicative of the possibility of glaucoma arising (*vide* p. 285), atropine should be avoided and the eye should be examined daily for a time. Atropine should not be used as a routine measure: it is generally unnecessary, and it always involves prolonged absence from work and consequent economic loss. In every case parolein is instilled, and the eye is kept bandaged for a day, and boric lotion is ordered. If ulceration occurs it is treated in the appropriate manner (*vide* p. 204). Special attention should be given to particles of stone, which show a greater tendency than steel,

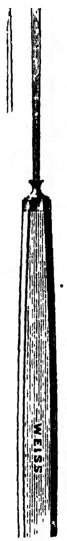


FIG. 225.—Spud for removing foreign bodies from the cornea.

&c., to cause ulceration, probably because steel particles are often hot, and therefore sterile, when they enter the eye.

Occasionally sharp steel and other particles penetrate deeply into the cornea, without, however, perforating. The efforts made to remove them may push them still deeper or even into the anterior chamber. When such an accident is feared, special precautions must be adopted. If the particle is steel and a large magnet, *e.g.*, Haab's or Mellinger's, is available, this method should be tried (*vide* p. 460); it is often necessary to incise the cornea overlying the foreign body. This method may fail, the particle being so small that an insufficient number of lines of force pass through it. In these cases, or when the particle is non-magnetisable, a broad needle should be passed into the anterior chamber and pressed against the back of the cornea while the foreign body is being removed with a needle. Usher has invented a spatula, curved on its anterior surface to fit the back of the cornea, for this purpose; it is introduced into the anterior chamber through a keratome incision. If the foreign body escapes into the anterior chamber it must be removed by other methods (*vide* p. 457).

Prophylactic Measures. Foreign bodies in the eye are extremely common in industrial workers, especially in grinding tools, lathe work, &c. Apart from the danger to the sight of the worker, they are a source of great economic loss from loss of time, compensation, &c. They could in most cases be entirely prevented by the use of protective goggles, but it has hitherto been found impracticable to enforce this measure among British workmen. Every attempt should be made by the provision of comfortable goggles and by educative means, such as "Safety First" notices and lectures by welfare officials, to point out the dangers and to encourage the workmen to use goggles.

Burns and Injuries by Caustics. Burns by hot water or steam, hot ashes, exploding powder, molten metal, &c., and injuries by caustics, such as lime, usually from fresh mortar or whitewash, strong acids and alkalies, &c., endanger the eye chiefly in two ways, viz., by injuring the cornea and by producing symblepharon. Strong ammonia is particularly harmful, causing necrosis of the cornea; hydrochloric acid (spirits of salt) much less so. Many eyes have been lost through cutting open golf balls: the central core often contains caustics (barium sulphate, caustic soda, etc.) which spurt into the eye. Immediately after the accident there is intense conjunctivitis and chemosis, but the cornea looks clear; in this state it is difficult to be certain of the extent of

the injury. A drop of fluorescein solution will reveal the extent of the area denuded of epithelium. Prognosis should therefore be guarded, care being taken to impress upon the patient the gravity of the injury and the necessity for constant supervision. In the worst cases the cornea is dull or opaque. In the succeeding days an eschar forms and is thrown off. This is followed by granulation of the injured conjunctiva and frequently by ulceration of the cornea. The corneal condition must be treated like a corneal ulcer (*vide* p. 204). In bad lime burns, &c., the whole cornea may be destroyed; perforation takes place, and the eye shrinks. In less severe cases a dense leucoma forms, porcelain-like in lime burns, and sight is lost. The chief danger derived from the condition of the conjunctiva is that of adhesion of the lid to the globe. It is most likely to occur with the lower lid, the caustic acting principally upon the lower fornix, which is obliterated by organisation of the granulation tissue. The symblepharon thus produced impedes the movements of the globe and may even interfere with its nutrition. Every precaution must be adopted to prevent its occurrence.

Treatment. In the earliest stages of injury by caustics the excess of deleterious material must be removed. Acids may be neutralised by dilute alkalies (lotio sodii bicarbonatis, 3 per cent.) and alkalies by weak acids (lotio acidi borici) or milk. Particles of lime must be perseveringly picked out with forceps, after previous application of pantocain: irrigation with 10 per cent. solution of neutral ammonium tartrate is painful but undoubtedly diminishes scarring in lime burns. A few drops of ol. ricini or parolein may be instilled.

To prevent symblepharon a glass rod is well coated with vaseline or boric acid ointment, or if the cornea is involved atropine ointment, and the point is swept round the upper and lower fornices, so that they are well packed with ointment. In severe cases cold compresses should be applied, and the patient put to bed. In the succeeding days, if symblepharon still threatens, the treatment with ointment and the glass rod is repeated once or more times daily according to the severity of the case.

Recently good results have been obtained by excising all damaged bulbar conjunctiva up to the limbus and down to the sclera. If the raw area thus left is small and there is enough conjunctiva available it may be covered by a conjunctival flap, but if it is large a graft of buccal mucous membrane is taken from inside the lower lip (or amnioplastin)

and sutured in position. Parolein is instilled, and the closed lids covered by a layer of tulle gras, a pad wrung out in saline and a crêpe bandage.

A contact glass coated with sterile vaseline inserted over the cornea and conjunctiva assists in preventing symblepharon. If symblepharon occurs it must be suitably treated (*vide* p. 637), but prevention in this case is easier than cure. If the actual fornix is denuded of epithelium it may be impossible to prevent symblepharon.

Contusions by Blunt Instruments

Injuries by blunt instruments vary in severity from a simple corneal abrasion to rupture of the globe. There is no part of the eye which may not be so injured by contusion as seriously to diminish vision. Moreover, in some cases the changes are progressive, so that *in all cases a very guarded prognosis should be given.* The various conditions which may follow contusion will be briefly enumerated.

Cornea. A simple *abrasion* may be caused. It is recognised by distortion of the corneal reflex and by the use of fluorescein (*vide* p. 87). There is much pain, like that due to the presence of a foreign body, increased on moving the lids, much lacrymation and reflex blepharospasm. It may become infected and give rise to a corneal ulcer, especially if a mucocele is present (*vide* p. 653). In the simple cases the use of a lotion, *e.g.*, boric acid, boric ointment to prevent the lids from sticking together, and a pad and bandage for a few days suffices (cf. p. 434). Ulceration must be treated suitably (*vide* p. 204).

Recurrent Erosion (*Syn.—Recurrent Traumatic Keratalgia*) is particularly liable to occur after scratches with babies' finger-nails. The abrasion, however produced, usually heals quickly, but is followed some days, weeks, or even months later, by acute pain and lacrymation, generally on first opening the eyes in the morning. If the cornea is then stained with fluorescein an abrasion will be found, usually at the original site, but sometimes elsewhere, or there may be a vesicle or group of vesicles. The attack rapidly passes off with appropriate treatment, but often recurs again and again. There is no doubt that in these cases the epithelium is abnormally loosely attached to Bowman's membrane, and is liable to be torn off by the lid on waking. Such looseness of epithelium and formation of vesicles is characteristic of lesions of the fifth nerve (*vide* pp. 227, 229), and it is probable that recurrent erosion is due to this cause, though the actual rationale is unknown. Early attacks should be treated in the

same manner as a simple abrasion, boric ointment being plentifully applied at night. Instillation of 1 per cent. pantocain relieves the pain and seems to have a good effect on the epithelium. If the attacks are repeated the spots should be curetted and touched with strong alcoholic solution of iodine (*vide* p. 230) or pure carbolic acid (*vide* p. 218): mild application of X-rays has been found beneficial (Greeves) (*vide* p. 234).

Deep opacity may be found, usually in the form of delicate grey striæ interlacing in different directions. They are due to accumulation of lymph in the interlamellar spaces, occasionally to wrinkling of Descemet's membrane (*vide* p. 248). They generally clear up without leaving a permanent opacity.

Examination with the slit-lamp has shown that all contusions of the eyeball are followed by the deposition of fine granules of uveal pigment on the posterior surface of the cornea. There may be ruptures in Descemet's membrane; owing to its elasticity the edges are rolled over.

Blood-staining of the cornea occasionally results from contusion which has caused hæmorrhage into the anterior chamber. Probably in all the cases the tension of the eye is raised in the early stages. The whole cornea is at first stained, the colour varying according to the duration of the condition. It may be reddish-brown, or greenish. In the latter case the condition nearly simulates dislocation of the clear lens into the anterior chamber (*vide* p. 441). The cornea gradually and very slowly clears from the periphery towards the centre, the whole process taking two years or more. Microscopically there are myriads of minute, highly refracting rods packed in the lamellæ of the substantia propria, and sometimes round granules of pigment in the corneal corpuscles. These are derivatives of hæmoglobin, which may or may not contain iron. They are probably removed by the phagocytic action of leucocytes—a slow process. In the absence of other cause of defective vision sight may eventually be completely restored.

Rupture of the cornea is very rare. Descemet's membrane may be ruptured alone. In complete rupture an attempt may be made to save the eye by suturing the cornea with a special needle (Fig. 271).

Sclerotic. *Rupture of the globe* is generally due to it being suddenly and violently forced against the orbital walls. It is often due to falls upon some projecting object, such as a knob or a key in a door, and has been frequently caused in country districts by a blow from a cow's horn. The force is usually applied from the direction down and out, where the eyeball is

least protected by the orbital margin; the eye is forced into contact with the pulley of the superior oblique muscle. The sclerotic gives way up and in at its weakest part, viz., in the neighbourhood of the canal of Schlemm. The wound is oblique, being farther forwards internally than externally, where it appears more or less concentric with the corneal margin and about 3 mm. behind it. The conjunctiva is often intact, but there are always severe injuries to other parts of the eye. The iris is generally prolapsed or torn away (iridodialysis) or retroflexed (*vide* p. 440). The lens may be expelled from the eye or escape under the conjunctiva (subconjunctival dislocation of the lens) or be forced back into the vitreous, making the anterior chamber deep. The anterior chamber contains blood (hyphæma), and there may be hæmorrhage into the vitreous. Detachment of the retina may occur, with or without subretinal or subchoroidal hæmorrhage. The eye usually shrinks and is lost.

Treatment. The eye must be carefully examined with lid retractors, under an anæsthetic if necessary. In severe cases nothing remains but to excise the collapsed globe. In less severe cases, without extrusion of the contents of the globe, atropine may be instilled, cold compresses applied, and the patient kept in bed. Sometimes good results follow suture of the rupture. If the rupture involves the periphery of the cornea the iris alone may be prolapsed. It is then a good plan to insert the sutures in the sclerotic without tying them, before excising the prolapse; they are then tied.

In subconjunctival dislocation of the lens it would seem a natural procedure to open the conjunctiva and let out the lens. This is, however, contraindicated in the early stage. It must be remembered that there is an opening directly into the vitreous, and that such a procedure will almost inevitably involve escape of vitreous, and possibly panophthalmitis. Atropine should be instilled and cold compresses applied. The lens will gradually become absorbed, but no harm will accrue if the remnants are removed after the scleral rupture has healed.

Iris. Most injuries to the iris caused by contusion are due to sudden incurving of the cornea, whereby the aqueous is forced back against the iris and lens.

Traumatic mydriasis may follow a contusion. The pupil is large and immobile, and usually remains moderately dilated permanently. It is due probably to paralysis of the motor nerve fibres, which may be stretched or torn in their passage

through the ciliary body. There are minute ruptures in the pupillary margin, but these do not account for the immobility. There is usually also paralysis of accommodation. *Traumatic miosis* is rarer, and results from less severe injuries; it usually passes off. *Radiating lacerations* of the iris, sometimes extending to the ciliary margin, are rare (Fig. 226). *Iridodialysis* is commoner (Fig. 227). The iris is torn away from its ciliary attachment for a variable distance. On inspection a black biconvex area is seen at the periphery, and the pupillary edge bulges slightly inwards. With the ophthalmoscopic mirror a reflex can be obtained through the peripheral gap, and the fibres of suspensory ligament and edge of the lens may be visible. Uniocular diplopia may be produced by this injury. In

FIG. 226. — Lacerations of the pupillary margin of the iris and dislocation of the lens, following a blow. (From a drawing by Holmes Spicer.)

FIG. 227.—Iridodialysis following a blow. (After Nettleship.)

extensive iridodialysis the detached portion of the iris may be completely rotated, so that the pigmented back of the iris faces forwards (*anteflexion of the iris*). The iris never becomes re-attached, but iridodialysis, apart from other injury, rarely causes serious results. In *traumatic aniridia* or *irideremia* the iris is completely torn away from its ciliary attachment, contracts into a minute ball, and sinks to the bottom of the anterior chamber, where it may be impossible to see it. Rarely the same appearance is caused by *total inversion* or *retroflexion* of the iris, the whole iris being doubled back into the ciliary region out of sight. More commonly inversion is partial, so that the appearance of a coloboma (*q.v.*) is obtained, but the fibres of the suspensory ligament cannot be seen. In all these cases there is usually hyphæma; and other injuries, such as partial dislocation of the lens and so on, may be present.

The *treatment* consists in rest and the application of cold compresses. Atropine should be instilled in iridodialysis, but not in ruptures of the iris. If there is also subluxation of the lens neither mydriatic nor miotic should be used.

Ciliary Body. Contusions may cause diminution in the amplitude or loss of accommodation owing to paralysis of the ciliary muscle. The ciliary body may be involved in rupture of the globe (rupture, prolapse, &c.), and plastic cyclitis may be induced. Hypotony or low intraocular pressure may follow a blow, probably through interference with the functions of the ciliary body. It may persist for a long time, and be followed by degenerative changes in the lens, and shrinking of the globe.

Lens. Subconjunctival *dislocation* of the lens has already been described. The same mechanism which produces the various injuries of the iris may cause the lens to be forced back into the vitreous. The suspensory ligament is then ruptured. The rupture may be complete or partial. When complete the lens may sink to the bottom of the vitreous chamber, where it may be visible as a yellowish mass; occasionally it remains clear and cannot be seen. Partial rupture of the suspensory ligament occurs with subluxation of the lens, which may be displaced laterally with or without some degree of rotation. This leads to irregularity in the depth of the anterior chamber, which is deeper in the part unsupported by the lens. With the pupil dilated the edge of the lens may be seen, as a grey convex line by oblique illumination, but more readily and unmistakably as a black line with the ophthalmoscopic mirror (*vide* p. 110). The want of support to the iris causes tremulousness (iridodonesis) on the slightest movement of the eye, a tremulousness which is limited to the unsupported part.

Blows upon the eye less directly from before backwards occasionally cause dislocation of the lens into the anterior chamber. It rarely occurs with a lens of normal size, but not infrequently by quite trivial injury when the lens is shrunken. The clear lens in the anterior chamber is not always easily recognised, but it does not long remain clear, and diagnosis is then easy. It is more globular than normal owing to its freedom from the restraint of the suspensory ligament. When still clear it looks like a globule of oil in the anterior chamber. With oblique illumination it has a golden rim, due to total reflection of the light; this is the exact opposite of the total reflection when the edge of the lens is seen with the mirror, the light being then totally reflected away from the observer's

eye. The lens in the anterior chamber causes spasm of the sphincter iridis, which may occur at the moment when it is passing through the pupil. Intense iridocyclitis or secondary glaucoma is then set up. In most cases of dislocation forwards the lens is shrunken, and the suspensory ligament has become partially absorbed; dislocation into the anterior chamber may occur in these cases spontaneously, without any contusion. Unless the lens is very small, extreme irritation is set up by its presence in the anterior chamber, and the eye is lost if it is allowed to remain there.

Dislocation of the lens always causes considerable disturbance of vision. In partial rupture of the suspensory ligament there is astigmatism which is much increased by tilting of the lens. The slackening of the suspensory ligament causes increased curvature and myopia, which may be more than compensated by backward displacement. In total dislocation into the vitreous the effect is that of the old cataract operation of couching; the pupillary area is aphakic, the refraction is highly hypermetropic, requiring cataract glasses for its correction. Vision usually deteriorates gradually.

If the lens is displaced so much laterally that the edge crosses the pupil uniocular diplopia is present. Through the aphakic area of the pupil the eye is highly hypermetropic, through the phakic portion it may be myopic, in addition to which the periphery of the lens acts as a prism. Ophthalmoscopic examination under these conditions shows two images of the disc by the indirect method, differing considerably in size. By the direct method the fundus may be observed through the phakic or through the aphakic portion of the pupil, different lenses being required to correct the refraction in the two cases.

Subluxation of the lens may occur as a congenital condition (ectopia lentis) (*vide* p. 329).

Besides the immediate consequences of dislocation of the lens, very serious remote effects may follow. In subluxation the lens is very liable to become opaque, owing to malnutrition. The pressure of the edge of the lens on the back of the iris and on the ciliary body often sets up severe iridocyclitis, which may lead to the loss of the eye, and even endanger the other by sympathetic ophthalmia (*q.v.*). Secondary glaucoma is a very frequent sequel (*vide* p. 282).

Treatment. In dislocation forwards or subluxation miotics must be avoided, since the contraction of the iris behind the lens may actually induce secondary glaucoma. In the absence of irritation vision may be improved in total luxation into

the vitreous and in subluxation by suitable glasses. In the latter case it is usually impossible to correct the astigmatism, but sometimes the aphakic part of the pupil can be used. If iridocyclitis or secondary glaucoma is present the lens should be extracted if it is possible. It is imperative when the lens is in the anterior chamber. In all cases it is unusually difficult. There is always a considerable rupture of the suspensory ligament, so that vitreous presents as soon as the corneo-scleral section is completed, and the delivery of the lens has usually to be effected with the scoop, some vitreous being lost (*vide* p. 496). If extraction is impossible an iridectomy or trephining may improve matters, but more usually fails. If the eye is blind and painful it should be excised. For the treatment of subconjunctival dislocation see p. 439.

Besides dislocation of the lens, *concussion cataract* occasionally follows a contusion. In most of these cases the capsule is ruptured, though the site of rupture, usually behind the equator, cannot be seen clinically. The lens gradually becomes opaque, a rosette-shaped opacity being usually first formed in the posterior cortex (Koby, Fig. 233). Rarely the cataract remains in this condition, but much more commonly it spreads throughout the cortex until the appearance of a mature cataract is found. More or less absorption takes place, but it may be permanently incomplete. The condition should be treated in the same manner as traumatic cataract.

In some cases a circular ring or disc of faint or stippled opacity is seen on the anterior surface of the lens (Vossius' ring). It usually has about the same diameter as the pupil, and has been attributed to the impress of the iris on the lens, produced by the force of the blow driving the cornea and iris backwards. This view is supported by the not infrequent presence of iris pigment on the lens capsule, but is opposed by the variation in the size of the disc and the occasional presence of a second concentric ring. The slit-lamp shows that the opacity is due to multitudes of brown amorphous granules lying on the capsule. It may form in the absence of hyphæma. Minute discrete subcapsular opacities may be seen after resorption of the pigment, which takes place very gradually.

Concussion cataract may also be caused by lightning and high tension electric discharges. It is possible that electrolytic changes play some part in these cataracts.

Vitreous. Hæmorrhage into the vitreous is the commonest effect produced in it by contusion. The vitreous chamber may

be filled with blood. In this case no reflex will be obtained with the ophthalmoscopic mirror. With oblique illumination a dull red hue may be seen, especially if the pupil is dilated. The blood may become almost completely absorbed, but cloudy opacities remain. In rare cases "retinitis" proliferans follows from organisation of the clot (*vide* p. 363).

Liquefaction of the vitreous and opacities in it may follow a blow owing to uveitis and defective nutrition, and without hæmorrhage.

Choroid. *Rupture of the choroid* occurs as the result of severe contusion. It has also been caused by a bullet passing through the orbit behind the eye. Immediately after the injury the view is obscured by extravasation of blood. When it has become absorbed the rupture, usually not far from the disc and concentric with it, is seen as a curved white streak over which the retinal vessels pass (Plate IX., Fig. 2). The retina may also be ruptured, but this is exceptional. The edges of the streak are pigmented in the later stages; in the earlier, remnants of blood may be seen. The white appearance is due to the sclerotic shining through. The rupture is generally to the outer side of the disc, and there are often two or three of different sizes, more or less concentric with each other. If the choroid is ruptured near the macula loss of central vision results. If the retina is ruptured also or becomes atrophied throughout its thickness, including the nerve-fibre layer, a large sector-shaped scotoma is produced. Simple ruptures of the choroid in which the macula is not involved cause little impairment of vision. The treatment consists of rest in bed until all extravasated blood is absorbed; atropine and dark glasses, and abstinence from reading, &c.

A contusion may cause choroidal hæmorrhage, which may be small, shown later by patches of choroido-retinal atrophy, or large, subretinal or subchoroidal. The latter can seldom be seen ophthalmoscopically, but are part of more extensive mischief.

Retina. It has already been pointed out that *detachment of the retina* (*q.v.*) is often due to contusion. *Rupture of the retina* with rupture of the choroid is rare in civil life, but was a common result of explosions during the War. *Hæmorrhages* into the retina occur; they are usually small, but large hæmorrhages into the vitreous (*vide supra*) are in part derived from retinal vessels.

Commotio retinæ is a frequent result of blows upon the eye. Instead of the normal bright red colour the retina shows a milk-

white cloudiness, usually near the papilla and posterior pole, and over a considerable area. It is probably due to œdema. It disappears after some days, and vision is usually restored to normal. In other cases, though vision may be good at first, central vision gradually diminishes, associated with development of pigmentary deposits at the macula (*vide infra*). Hence prognosis should be guarded in *all* cases of serious blows upon the eye.

Serious changes are apt to occur at the macula, and are easily overlooked immediately after the accident, or it may be impossible to obtain a good view. A "*hole*" may occur at the macula. It appears as a small circular or oval deep red patch, just as if a hole had been punched out. It is caused by cystic degeneration following œdema due to commotio retinæ, and in course of time a complete perforation of the retina develops. Similar ophthalmoscopic signs and pathological changes have been observed occasionally after iridocyclitis, and associated with arteriosclerosis, renal retinitis, and amaurotic family idiocy (*vide* p. 356). In other cases the macula looks deeper in colour than normal, and in the course of time it becomes pigmented. The spots of pigment are very fine, mostly aggregated near the fovea, with a few farther afield. This *pigmentation* is the sign of serious defect in central vision, which has a tendency to increase progressively.

Optic Nerve. The optic nerve is not infrequently injured in fractures of the base of the skull (*vide* p. 404). Injuries by sticks or knives, &c., penetrating the orbit are rare. Avulsion of the optic nerve is very rare in civil practice, but occurs in gunshot wounds of the orbit (*vide* p. 671).

Perforating Injuries

Perforating injuries may be caused by sharp instruments or by foreign bodies.

A wound with a sharp instrument may penetrate the cornea, the corneo-scleral junction or the sclerotic; it may pass in for a variable distance, wounding the iris, lens, &c., or pass through the eye.

Wounds of the Conjunctiva are common. They heal readily, but the process may be hastened, and the resulting adhesion to the sclerotic lessened by introducing one or more sutures. Polypoid masses of granulation tissue sometimes form on the surface; they should be snipped off with scissors after application of pantocain.

Wounds of the Cornea may be linear or lacerated. The

margins swell up and become cloudy through imbibition of fluid. This facilitates closure of the wound and restoration of the anterior chamber. If small and limited to the centre they heal well unless they become infected. The eye is irrigated with boric lotion or sterile saline, atropine instilled, penicillin ointment inserted, and a pad and bandage applied. A permanent dense opacity is left, and the contraction of the organising scar tissue causes irregular astigmatism. If the wound becomes infected it must be treated like a perforating ulcer.

The danger is greatly increased (1) if the wound is large, especially if it extends into the sclerotic, or (2) if the lens is also wounded. In the former case prolapse of the iris is almost certain to occur. The prolapsed iris should never be replaced, even if this is possible, since it may carry infection into the eye. It must be excised (*vide* p. 212), care being taken that the iris is quite free, though this is a counsel of perfection not always to be attained. Freeing of the iris is specially difficult with quasi-tangential wounds (*vide* Fig. 234 to the right). In such cases it is a good plan, if there is an anterior chamber, first to make a small keratome incision in the cornea on the opposite side to the wound, 3 mm. inside the limbus (Fig. 228). After the prolapse has been excised in the usual manner a repositor can be passed through this incision and swept over the surface of the iris (Goulden). This device is useful for freeing the iris from punctured wounds near the limbus, in which incarceration occurs, but is so small as to make excision through the original wound impossible. In some cases it is advisable to cover the wound with a conjunctival flap (*vide* p. 211). Atropine should always be instilled (never eserine).

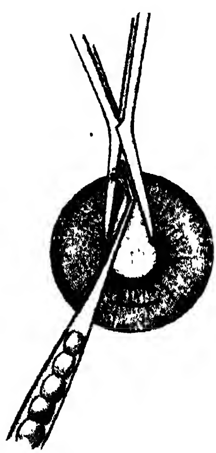


FIG. 228.

If in a few days it is found that there is an anterior synechia, this should usually be divided as soon as the wound is sufficiently healed to permit of the necessary procedures without re-opening it. If this is not done the traction on the iris will keep up irritation in the eye, as shown by ciliary injection, &c.,

and such eyes are liable to cause sympathetic ophthalmia. Moreover, a broad anterior synechia tends to bring about secondary glaucoma, or predispose to secondary infection and panophthalmitis.

Wound of the lens very greatly increases the gravity of the case, especially in children. It may escape notice at first, especially if the wound is small, *e.g.*, that caused by a needle or thorn. The lens swells and keeps the iris in contact with the cornea, so that re-formation of the anterior chamber is much delayed. If at length it re-forms the aqueous becomes filled with swollen lens fibres, which also irritate the iris. The swelling is greater and more rapid the younger the patient. Infection, which is one of the greatest dangers of all perforat-

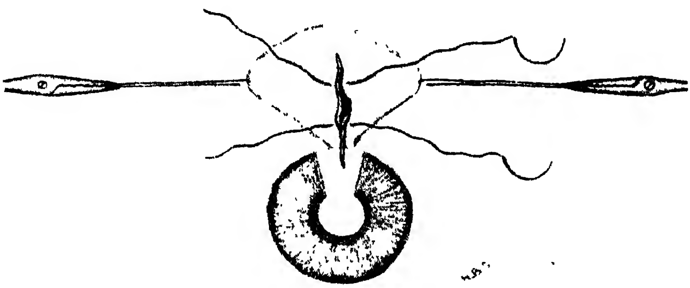


FIG. 229.

ing wounds, is particularly likely to occur in these cases. The excision of any prolapsed iris in such a manner as to free it completely from the wound is extremely difficult. The subsequent prolonged contact of the iris with the cornea facilitates the formation of broad adhesions of iris and often also of lens capsule which it may be found impossible to divide, or which, if divided, quickly re-form. Ciliary injection is kept up, and sympathetic ophthalmia is very liable to supervene if the eye is retained too long.

Occasionally in perforating wounds with a dirty implement pyogenic organisms are carried into the eye, multiply there and cause rapid necrosis of the whole cornea. In these cases a ring of deep infiltration appears in the cornea 2 or 3 mm. internal to and concentric with the corneo-scleral margin—so-called *ring abscess.* There is much chemosis of the conjunctiva and a greenish discharge. The organism has gener-

ally been found to be the bacillus pyocyaneus. Usually panophthalmitis is set up and the whole of the central part of the cornea is cast off. The only chance of saving such an eye is to do a paracentesis directly the infiltration is observed and to wash out the anterior chamber with pure penicillin solution (1,000 units per c.c.). This should be reinforced by subconjunctival injection of penicillin (*vide* p. 696).

Wounds of the corneo-sclera are particularly liable to set up sympathetic ophthalmia (*q.v.*). In them the ciliary body is injured directly, and may be prolapsed. There is the usual danger of infection, and if it occurs panophthalmitis is certain to follow on account of the ready communication with the vitreous. If this does not happen ciliary irritation is kept up. If suppuration occurs there is little hope of saving a useful eye, but there is little or no danger of sympathetic ophthalmitis.

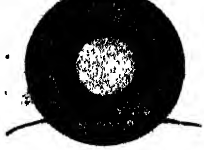


FIG. 230.

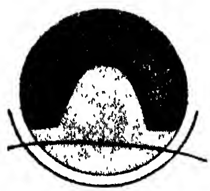


FIG. 231.

If the lens is wounded the chances of saving the eye are greatly diminished.

Treatment. An attempt should be made to save the eye. Under regional anæsthesia and akinesia (*vide* p. 472). the wound should be freely exposed as in Fig. 229. The edges of the scleral wound are carbolised and sutures inserted by an eyeless corneoscleral needle (Fig. 229). The sutures are looped ready to tie. Any prolapsed iris and ciliary body is drawn out of the wound and abscised and the scleral sutures are tied immediately so as to minimise loss of vitreous. Finally, the wound is covered with conjunctiva. Atropine and penicillin are instilled, the eye bandaged lightly, and the patient put to bed. Wounds involving the sclera on both sides (Fig. 230) are specially difficult to deal with. Before excision of the prolapse the conjunctiva is freed at the limbus (Fig. 231) and afterwards drawn over the wound by stitches inserted as in Fig. 232. Such a flap supports the cornea and helps to bring the edges of the wound in apposition, at the

same time assisting to protect the eye from intraocular infection.

If the eye does not quiet down in the course of a week or ten days, as shown by diminution of ciliary injection and cessation of photophobia and lacrymation, it should be excised. In the interval the cornea is examined most carefully each day by oblique illumination with the loupe for precipitates ("k.p."). If they are seen the eye should be excised. Similar care is devoted to the discovery of spots of "k.p." in the other eye, and if they should be found there excision of the injured eye is still more imperative at the earliest possible moment. If the eye quiets down quickly and there is no evidence of iridocyclitis in either eye the case will probably do well, but

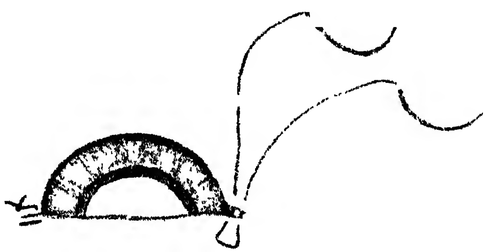


FIG. 232.

it should be kept under observation for a prolonged period (*vide* p. 464).

If there is much prolapse of vitreous as well as of iris and ciliary body, or if the lens is wounded, there is little probability of saving the eye. If it is almost certain that useful vision will be lost the risks of sympathetic should not be run, but the eye should be promptly excised.

Wounds of the sclerotic are not always easily recognised. The eye may have been wounded through the lid. The bruising and laceration of the lid may make examination of the eye difficult. The lid should be raised from the globe and drawn back with a Desmarres' retractor, under local anæsthesia. Even when the eye is examined the effusion of blood under the conjunctiva may render the diagnosis uncertain, still more the question of perforation. When perforation has

occurred, there is reduction in the intraocular tension. If the perforation is near the cornea, the anterior chamber is shallow or obliterated. If the wound is large, prolapse of some of the contents of the globe occurs. The uvea—iris, ciliary body, or choroid—are most easily recognised on account of their pigmentation. Very often the gelatinous vitreous can be seen hanging out of the wound. Hyphæma and vitreous hæmorrhage may be present with or without perforation.

Treatment. If the injury is so severe that there is no likelihood of recovery of useful vision, the eye should be excised. If there is a chance of useful vision, or if permission to excise is withheld, the sclerotic may be sutured and the sutures tied after excision of prolapsed vitreous, &c. Very small wounds do not require suturing, but the conjunctiva should be cleansed and stitched over them. Atropine and penicillin should be instilled, and both eyes bandaged. Complete rest in bed is imperative. Such eyes usually shrink, unless indeed panophthalmitis ensues.

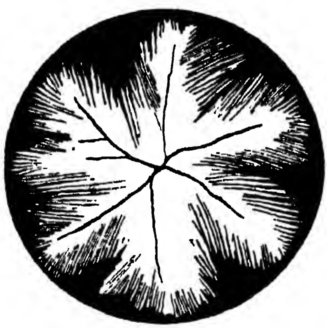


FIG. 233.—Traumatic posterior cortical cataract. (Koby.)

Wounds of the lens cause traumatic cataract. Usually the anterior capsule is wounded; when the posterior capsule is torn, the lymph in the vitreous acts in the same manner as the aqueous. A few hours after the injury the lens becomes cloudy in the vicinity of the wound owing to the action of the aqueous upon the fibres, causing them to swell. In most cases opacities rapidly form in the posterior cortex, irrespective of the exact site of the wound. They are at first feathery lines, which follow the natural distribution of the lens fibres. Later a rosette-like opacity is formed which gradually spreads until the whole cortex is opaque (Fig. 233). Flocculent grey masses protrude through the opening in the capsule, some of which become free and sink to the bottom of the anterior chamber. Sometimes the whole chamber is full of white flocculi, and the lens nucleus may escape entire. The masses are gradually dissolved by the aqueous, and pass out with it through the angle of the chamber. In this manner, in young patients, the whole lens, with the exception of the capsule, may be absorbed. Usually absorption ceases earlier through closure of the capsular wound.

The enclosed lens fibres become opaque, and discission is necessary to cause their complete absorption ; in adults, with a hard nucleus, extraction may be necessary (*vide* p. 490). The smaller the wound in the capsule, the sooner it is likely to close. In rare cases, with very small, though it may be deep, wounds, the opacity may remain limited to the site of injury, especially if the wound is quickly sealed up by a posterior synechia.

A certain amount of inflammatory reaction with ciliary injection is always set up, which may be excessive, even in the absence of infection. It will then be found that the intraocular tension is raised. The swelling of the lens forces the periphery of the iris against the cornea and secondary glaucoma supervenes. This tendency is increased by the difficulty with which the highly albuminous aqueous escapes from the eye, especially if the wound in the capsule is large and much of the lens substance has escaped into the anterior chamber. If the secondary glaucoma is not relieved, the sight will be lost by destruction of the optic nerve. The aqueous and swollen lens substance must therefore be let out by a curette evacuation (*vide* p. 488).

Traumatic cataract is deliberately induced in the operation of discission.

In the absence of secondary glaucoma, the condition is treated by rest in bed, atropine, and a bandage. It is of the utmost importance that the pupil should be kept well dilated. If it is not, adhesions will form between the iris and the lens capsule. The tendency to iritis is increased, a ring synechia may form, leading to a more serious form of secondary glaucoma, and in any case a subsequent needling will be made more difficult and dangerous. *Sterile* atropine ointment, 1 per cent., should be used three or four times daily. If the pupil does not dilate satisfactorily, hot bathings should be given every four hours, mydricain (*vide* p. 694) injected subconjunctivally, and medical diathermy applied once or twice a day : in the case of adults leeches are sometimes useful. It is usually necessary to needle the lens capsule in order to obtain an opening through which vision is made possible, and needling may have to be repeated (*cf.* Lamellar Cataract).

Perforating wounds with retention of foreign bodies have special features and dangers. Eyes containing foreign bodies are particularly liable to set up sympathetic ophthalmia. In cases of perforating wound, the question often arises whether a foreign body has been retained within the eye. The foreign bodies most likely to penetrate the eye and be retained are

minute chips of steel, stone, and particles of glass, lead pellets, copper percussion caps, less frequently spicules of wood. In chipping stone with an iron chisel, it is usually a chip of the chisel and not of the stone which enters the eye. Among war injuries penetration of the eye by fragments of the casing of rifle bullets, often containing nickel, of shells, &c., frequently occurs.

The *size and velocity* of the missile are of importance. If the foreign body is large so much damage is usually caused that the eye has to be removed. Very minute particles can, however, penetrate the cornea or sclerotic and lodge in the deeper parts of the eye. The velocity of these small particles must be very high, for the energy needed to penetrate the walls of the globe is considerable, and the relationship of energy to mass and velocity is given by the formula $E = \frac{1}{2} m v^2$. This is a point of more than academic interest. It has been shown that rifle bullets are sterilised by their rapid transit through the air. This fact may account to some extent for the sterility of minute intraocular foreign bodies, though not entirely; for they are not always sterile, and when they are it is often more probably due to being hot when emitted. Their irregularity of shape would render them less easily sterilised. *Cæteris paribus*, metallic foreign bodies appear to be more commonly sterile than those made of other substances.

The *nature of the foreign body* is very various and affects the diagnosis, pathological condition, treatment, and prognosis profoundly. As regards diagnosis, it is only rarely that we can see the foreign body in the eye or have indubitable proof of retention. In the absence of irrefragable evidence our surest test is skiagraphy, but this method has severe limitations. Relatively few substances are opaque to X-rays. Fortunately, by far the greatest number of intraocular foreign bodies are composed of iron or steel, which give a good shadow. The same applies to lead and metallic fragments from the casing of rifle bullets. Particles of glass often fail to reveal their presence in the skiagram, except heavy lead glass, of which some bottles are made. Very accurate localisation is necessary. Various methods are in use; Mackenzie Davidson's method is usually employed in England, Sweet's method, dependent upon the same principle, in America. When it is remembered that accuracy of localisation of the order of 1 mm. is essential and that inaccuracy of this amount may lead to the needless sacrifice of an eye, it

will be recognised how culpable is any carelessness in this respect.

The influence of the nature of the foreign body on the pathological condition set up in the eye is profound and varied. Thus, it was shown by Leber many years ago that copper causes suppuration even in the absence of pyogenic organisms, leucocytosis being set up by chemical action. Clinically, however, this result may certainly be delayed for a very considerable period, possibly owing to the copper—usually a fragment of a percussion cap—being surrounded by a wall of inflammatory material and encysted.

Copper in the lens may cause little reaction. This is further evidence in favour of the usual reaction being chemical, for such changes are very slow in the lens. Another fact supporting this explanation is that even in severe suppuration induced by copper within the eye the inflammation tends to cause shrinkage of the globe and not perforation. Not infrequently, however, the reaction is so severe as to cause expulsion of the foreign body from the eye. The pus formation retrogrades after a time and is not progressive. Only very rarely does a copper particle become encapsuled, with restoration of useful vision. Sympathetic ophthalmia is less likely to follow copper foreign bodies than others, probably owing to the intense reaction (Leber).

Iron is also dissolved by the tissue fluids and sets up the condition known as *siderosis bulbi.* The earliest clinical manifestation is the deposit of iron in the anterior capsular cells of the lens. These are not affected uniformly, but oval patches of the rusty deposit are arranged radially in a ring corresponding with the edge of the dilated pupil. This appearance is pathognomonic. At later stages the iris becomes characteristically stained, first greenish and later reddish-brown. The vision of these eyes, however little affected by the primary injury, gradually fails owing to degenerative changes in the retina and lens.

Siderosis bulbi (Bunge) has been exhaustively investigated. In a typical case brown granules are found in the corneal corpuscles, in the meshes of the ligamentum pectinatum iridis, on the inner surface of the ciliary body, and in the retina. The anterior layers of the iris are impregnated, and in addition to subcapsular deposits in the lens, the fibres are also stained. The retina shows complete degeneration, and Perls' micro-chemical reaction shows the whole retinal vascular system marked out by blue coloration. There is always intense blue coloration imme-

diately around the foreign body. The pigment epithelium of the ciliary processes, pars ciliaris retinæ, and retina, and sometimes the supporting tissues of the retina, show diffuse staining. The brown pigmented cells which give the blue reaction are found particularly in the angle of the anterior chamber and in the retina, less in the iris, and least in the choroid. They are not bleached by the ordinary methods for bleaching the normal pigment. Hæmorrhage associated with the injury introduces a complication, for it causes hæmatogenous pigmentation giving the iron reaction and distinguished with difficulty from the xenogenous pigmentation due to the foreign body.

The chemistry of siderosis bulbi is not yet fully understood. E. von Hippel says that the iron is dissolved by the carbon dioxide of the tissues and is fixed by cells which have a specific affinity for the metal; it then becomes oxidised. It has also been suggested that the iron is dissolved by acid phosphates in the intraocular fluid, or that iron may enter into solution in organic form as an albuminate or in combination with an organic acid. The brown precipitate in the tissues is almost certainly produced by oxidation, but it is not a simple oxide or hydroxide, as it is only very slightly soluble in oxalic acid (McMullen).

The characteristic ring of brown spots under the lens capsule is caused by deposition of iron in circumscribed aggregations of newly proliferated capsular epithelial cells. Leber showed experimentally that the introduction of a particle of iron into the vitreous causes extreme degeneration of the retina. Peculiar large granular cells are found which are derived for the most part from the retinal pigment epithelium.

Metals, other than iron and copper—such as lead, zinc, gold, silver—appear to cause little chemical reaction and usually remain quiescent, becoming more or less thoroughly encapsuled according to their position. Lead becomes coated with the carbonate.

Stone is chiefly dangerous from pyogenic infection, but chemical changes also occur, varying with the nature of the stone.

Glass and porcelain may cause remarkably little reaction, but iridocyclitis and disorganisation of the eye usually occur eventually.

With regard to wood, apart from infection, the most characteristic feature is the local irritation produced, resulting in the formation of dense granulation tissue, studded with so-called foreign body giant cells.

Eyelashes may be carried into the anterior chamber in perforating wounds of the cornea, whether accidental or

operative; and caterpillar hairs may penetrate the globe (*vide* p. 188).

More important numerically than the chemical changes are those due to infection, and though these are not, strictly speaking, due to the nature of the foreign body, it is undoubtedly true that certain types of foreign body are more apt to give rise to suppuration than others. Much may be learnt in this connection from the analogy of hypopyon ulcer. The common hypopyon ulcer is due to pure or mixed infection with pneumococci, and it is notorious that it is more likely to be caused by fragments of stone, wounds with the leaves of plants or twigs of trees and so on, than by steel or other foreign bodies. Moreover, it admirably exemplifies the *rôle* played in such infections by the resistance of the tissues, for the patients are usually either old and debilitated or alcoholic. With regard to intraocular infections, it is to be borne in mind that the lens substance and the vitreous form excellent culture media, and further, that even saprophytic organisms, like bacillus subtilis, are capable of setting up a suppurative inflammation in the eye. Probably the commonest pyogenic organism in the interior of the eye is, however, the pneumococcus.

The foreign body may pass through the cornea or the sclerotic. The wound of entry may be extremely minute. The patient may even be unaware that a foreign body has penetrated the eye. If it has passed through the cornea, the minute wound or scar can always be found by careful examination with oblique illumination and a loupe. It may escape detection in the sclerotic.

The foreign body may be retained in the anterior chamber. Here it may fall to the bottom of the chamber, and if very small be hidden by the sclerotic. It is generally, however, caught in the iris, and can be recognised with a loupe. A piece of glass in the anterior chamber is exceptionally difficult to see, on account of its refractive index differing so little from that of the surrounding media.

The foreign body may pass into or through the lens, either by way of the iris or of the pupil. In each case a traumatic cataract is produced, which undergoes the usual changes (*vide* p. 450). If the particle has passed through the iris there will be a hole in this structure. If the case is seen very early or very late, the hole looks black by oblique illumination, but shows a red reflex when illuminated by the ophthalmoscopic mirror. In the intermediate stage the cataractous

lens behind the hole prevents a red reflex from being seen. A hole in the iris is of great diagnostic significance, since it rarely occurs except as the result of perforation by a foreign body. The foreign body may be visible in the lens, either before or after dilatation of the pupil. It is possible for a foreign body to pass through the iris and through the circumlental space without wounding the lens.

The foreign body may be retained in the vitreous. Access to the vitreous by the foreign body may be given by various routes: through the cornea, pupil and lens; through the cornea, iris and lens; through the cornea, iris and zonule; or through the sclerotic. Hildebrand found these four routes represented in forty-three cases by 6, 16, 6, and 15 respectively. The particle may pass quite through the globe into the orbit, remain near the site of entry, become imbedded in the opposite wall, rebound from it, or be suspended in the vitreous. In the latter case it eventually sinks to the bottom of the vitreous chamber owing to degenerative changes in the humour, which lead to liquefaction, partial or complete. Sometimes air is carried in and appears as bubbles in the vitreous; these rapidly become absorbed. If the particle is small, the lens clear, and there has been little hæmorrhage, the body may be seen ophthalmoscopically in the vitreous or retina. The track through the vitreous looks like a grey line. The foreign body, generally black, and often with a metallic lustre, is surrounded by white exudate and red blood-clot. If the particle has been long *in situ* it may become more or less encapsuled, a small white area of fibrous tissue being seen with dense masses of black pigment in and around it. Fine pigmentary disturbance at the macula may follow, irrespective of actual injury to this region, and indeed merely as the result of concussion. More extensive degenerative processes also occur in the retina, which may become detached. Encapsulation is often rapid with iron, and useful vision may persist for an indefinite time. Particles more than 1—2 mm. in size are almost certain to lead to the destruction of the eye. In the absence of sepsis, siderosis bulbi is the almost inevitable cause of destruction. An encapsuled foreign body may become free after a long period of quiescence. The encapsulation of foreign bodies in the retina depends largely upon their asepsis. The amount of cicatricial tissue formed in the early stages is inversely proportional to the amount of necrosis, which depends chiefly upon bacterial invasion, though mechanical injury and chemical action must also be taken into account.

Retinal degeneration attacks the macula or is generalised. In the former group yellowish-white spots appear in the region of the fovea, and pigmentation may also occur. Serious disturbance of vision results, and is not recovered from. Generalised retinal degeneration takes the form of pigmentation, resembling that of retinitis pigmentosa, and may be preceded by night-blindness.

The prognosis is always bad. It is least bad if the foreign body is in the anterior chamber and the lens is not wounded. The eye may be saved if it is in the lens, especially in young people, whose lenses are capable of becoming completely absorbed. The prognosis is far better when the lens is not wounded, and this is still more markedly the case the younger the patient. With a small foreign body the wound of entry is so small that prolapse of iris rarely occurs, but if the lens is wounded, and more particularly if the patient is young, so that the swelling of the lens is excessive, the iris is pressed forwards against the cornea, anterior synechia is readily formed, and the obliteration of the angle of the anterior chamber easily leads to secondary glaucoma. If the iris is incarcerated in the wound in these cases the division of the synechia presents considerable technical difficulties, the iris and ciliary body are kept in a state of irritation, and the dangers of sympathetic ophthalmia are greatly enhanced. The satisfactory evacuation of the lens substance is much more difficult than in ordinary discission of the lens, so that the continued apposition of the wounded and inflamed iris to the cornea is scarcely to be avoided. Moreover, in my opinion, children are very decidedly more susceptible to sympathetic ophthalmia than adults.

Though the dangers of wound of the lens are diminished in older patients, there is no doubt that the prognosis is rendered graver by this complication. However satisfactory the result may be from the purely surgical point of view, an aphakic eye is decidedly less useful than one which retains a normal lens.

Treatment. It is a rule that the foreign body should be removed. There are rare exceptions to this rule, more particularly if (1) the foreign body was probably sterile; (2) little damage has been done to vision; and (3) the process of removal will almost inevitably destroy sight. These conditions are most often fulfilled in the case of minute foreign bodies in the retina.

Magnetisable foreign bodies are more easily removed than

others, since the small or the large electro-magnet can be brought to bear upon them.

A chip of steel free in the anterior chamber is removed in the following manner by the hand magnet (Fig. 235).

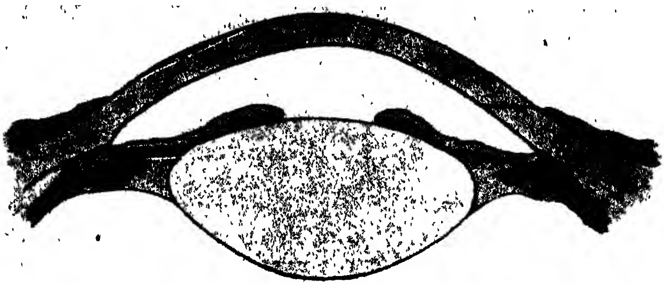


FIG. 234.

Retro-ocular anæsthesia or intravenous pentothal sodium should be used owing to the iritis already present. After washing out the eye a keratome incision is made above, 3 mm. from the limbus. The keratome is pushed straight on until the incision is of the required size, remembering that

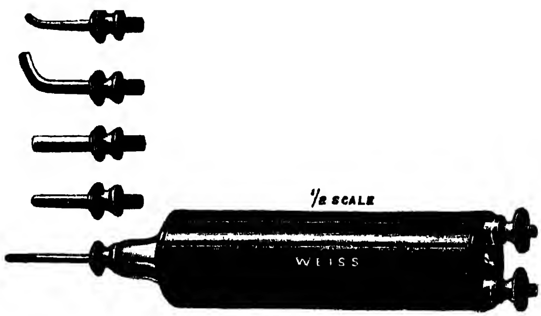


FIG. 235.—Small magnet, with poles of various shapes.

the internal wound in the cornea is smaller than the external. The keratome is removed moderately quickly but smoothly, so as to prevent loss of aqueous. The pole of the small magnet is then placed over the foreign body, outside the cornea, and moved towards the wound, thus dragging the particle along the back of the cornea. The posterior lip of the wound is then depressed with the pole and the foreign body is drawn

towards it, out of the wound. If much aqueous is lost it is more likely to become entangled in the iris, and considerable difficulty may be experienced in freeing it. It is wise to wait 20 minutes for the anterior chamber to re-form before removing the foreign body, and it may be necessary to pass a flat pole into the chamber. All the preparations for dealing with a prolapse of iris must be made (*vide* p. 212).

A foreign body on the iris may sometimes be removed with iris forceps through a similar incision. If it is entangled in the iris tissue, the iris must be drawn out of the wound and the part containing the foreign body excised with de Wecker's scissors, as in the operation for iridectomy (*vide* p. 475).

If a non-magnetic foreign body lies on the iris in the angle of the anterior chamber it is impossible to get at it with forceps by an ordinary keratome incision immediately over it, such as the corneal incision to the right in Fig. 234. The incision should be made at 3 mm. inside the limbus in the quadrant of the cornea lying over the foreign body, the point of the keratome being directed straight at the foreign body (Fig. 234 to the left). The foreign body can then be lifted out with toothless forceps, and the risk of prolapse of iris is also minimised (Goulden).

If the foreign body is in the lens, a few days should be allowed to elapse for the aqueous to act upon the lens fibres. A curette evacuation (*vide* p. 488) is then performed and the foreign body will probably be evacuated with the lens matter, or it may be removed by forceps, or if magnetisable, by the small magnet. In a young subject it may be advisable to increase the opening in the capsule by discission a few days before doing a curette evacuation. In these cases a subsequent needling will often be required to obtain a clear opening in the pupillary area of the capsule. It should be done in exactly the same manner as after discission for lamellar cataract. If the patient's other eye is normal he will not be able to wear the correcting glass for the aphakic eye, but his field of vision will be increased on this side. A more cogent reason for operating is found in the fact that it is easier to obtain a clear opening at this period than after months or years, when the capsule will have become thickened and very difficult to divide. In elderly patients it may be necessary to extract the lens by the operation for extraction of senile cataract (*vide* p. 490), but the large amount of soft lens matter will increase the dangers of the operation.

If the foreign body is in the vitreous or retina it is practically impossible to remove it without very seriously damaging the eye unless it is magnetisable. The treatment then lies between leaving it alone and watching the case, or excising the eye on account of the damage done or the danger of sympathetic.

If the substance is iron it may be possible to remove it with the large magnet (Haab's Giant Magnet or Mellinger's Ring Magnet). This will be facilitated by knowing its position, either by direct observation or by skiagraphy; some hint may be obtained from the position of the wound of entry and the probable direction in which the foreign body was travelling. If the patient is seen immediately after the accident it is best to use the large magnet at once, without waiting for a skiagram. The longer the foreign body is left the more firmly it becomes imbedded in exudates or fibrous tissue, and the less the probability of its successful removal.

The patient, whose pupil should be fully dilated, is seated in front of the magnet if Haab's instrument is used (Fig. 236). The eye is brought close to the magnet, the cornea touching the pole. This is important, since if there is a large piece of iron in the eye and the cornea is not in contact with the pole, the whole eye may be drawn forward out of the socket when the current is turned on. Moreover, the direction of the core of the magnet should coincide as nearly as possible with the direction of entry of the foreign body. The current is then turned on: some pain is usually felt when a piece of steel is present within the eye. If the operation is successful the foreign body comes forward into the posterior chamber. It may be necessary to turn the current on and off several times before this happens. If the particle is small and deeply imbedded the operation is likely to fail; hence it should be performed as soon as possible after the injury, before the chip has become firmly encapsuled or imbedded in exudates. The foreign body generally passes round the lens, not through it. It will be seen bulging the iris forwards. The current should then be turned off and the eye adjusted so that the particle will be drawn towards the pupil (Fig. 237). When it has fallen into the anterior chamber the patient is transferred to the operating table and the foreign body removed with the small magnet in the manner already described.

In some cases when the foreign body has been accurately localised and the large magnet has failed to bring it forward it can be removed by the small magnet introduced through a

scleral incision made by surgical diathermy as nearly as possible over the site. This posterior route is favoured by some operators, and is to be preferred in certain cases in which the foreign body is accurately localised.

The immediate effect of extraction of foreign bodies with the large magnet is often good, but irreparable damage is often done to the eye. The tracks through the vitreous often become filled with fibrous tissue. As this organises and contracts the retina is pulled up, and total detachment destroys vision. Or more severe iridocyclitis may be set up and the eye shrinks.

Fig. 236.—Removal of intraocular foreign body by Haab magnet: first stage. Drawing the foreign body into the posterior chamber, the eye looking forwards. (Goulden & Whiting.)

Fig. 237.—Removal of intraocular foreign body by Haab magnet: second stage. Drawing the foreign body across the posterior chamber from behind the iris. (Goulden & Whiting.)

The ring magnet has the advantage that the patient lies upon the operating table throughout. When a current passes round a solenoid a magnetic field is generated, its greatest saturation being in the central axis of the solenoid. The ring is placed over the patient's head, which is arranged so that the affected eye is as near the centre as possible. When the current is turned on every steel instrument placed within the ring becomes a magnet, the force varying with the mass of metal in the instrument and

its position in the ring. The foreign body is drawn forwards, as already described, by means of rods of soft iron of various sizes. The smallest should be used first, and the largest should be firmly grasped, otherwise they will be dragged out of the hand and thus do damage. As soon as the foreign body is seen to bulge the iris forwards the position of the rod is altered so as to draw it through the pupil. It may then be withdrawn by the small magnet or a keratome incision can be made, and the smallest rod or a steel spatula can be used in the same manner as the hand magnet.

Panophthalmitis

Panophthalmitis is generally caused by infected wounds. whether accidental or the result of operations, and ulcers. Less frequently it is metastatic, accompanying pyæmia and puerperal fever, meningitis, and orbital cellulitis (*vide* p. 343). In the exogenous form the vitreous is usually first affected, organisms grow in it as in a culture medium, and purulent cyclitis, retinitis, and choroiditis are set up (Fig. 238). In most cases the deeper parts of the vitreous are infected. In simple prolapse through a scleral wound the vitreous offers remarkable resistance to infection (W. A. Gray). In the endogenous forms there may be a septic embolism of a retinal artery or the choroid may be first affected. In this group it may be bilateral.

In both forms there is rise of temperature, headache, drowsiness, and sometimes vomiting. In the exogenous forms the edges of the wound become yellow and necrotic, hypopyon appears, there is great chemosis, with intense ciliary and conjunctival congestion, and the lids are swollen and red. There is severe pain in the eye, due at first to iritis, later to increased tension. The vitreous becomes purulent, as shown by a yellow reflex by oblique illumination. The anterior chamber soon becomes full of pus, and the cornea cloudy and yellow ; ring infiltration may occur (*vide* p. 447). There may be exophthalmos and limitation of movement of the globe, due to extension of the inflammation to Tenon's capsule. In the metastatic cases rapid failure of vision, a yellow reflex, and hypopyon are found in the early stages.

If the case is left to take its course the pus bursts through, usually just behind the limbus. The pain subsides, and after prolonged suppuration the eyeball shrinks. The prognosis is bad, the eye being almost invariably lost. The condition is not likely to set up sympathetic ophthalmia.

The pneumococcus is responsible for most cases of panoph-

thalmitis, but it is also caused by staphylococci, streptococci, bacillus coli, bacillus pyocyaneus (*vide* p. 448), clostridium Welchii (gas gangrene), and even by saprophytic organisms such as bacillus subtilis.

Treatment. In the early stages after operation wounds an attempt must be made to stay the process. The edges of the wound are cauterised with pure carbolic acid or the actual cautery. Penicillin treatment should be used to its fullest extent, 10,000 units of the *pure* salt being injected subcon-

FIG. 238.—Section of the retina and choroid in panophthalmitis (× 60), following a perforating wound.

junctivally every 3 or 4 hours, and in desperate cases an intraocular injection being made into the anterior chamber or even the vitreous (*vide* p. 697). Hot bathings, medical diathermy and leeches are applied, and atropine instilled. Sulphonamides should be given in full doses (*vide* p. 695).

As soon as it is evident that the eye cannot be saved it should be excised. This should not be left too long, and great care should be taken that no undue pressure is put upon the eye. If pus escapes there is danger of purulent meningitis being set up, the patient's life being thereby endangered. If there is any risk of this occurring the globe should be eviscerated

by excising the cornea and scooping out all the intraocular contents, special care being taken to leave no uveal tissue behind. The interior of the sclerotic is then swabbed out with perchloride lotion (1 in 2,000) and the conjunctival sac irrigated with a large quantity of weaker lotion (1 in 5,000). Most of the sclerotic may be excised, but a collar of it should be left around the optic nerve.

Sympathetic Ophthalmia

Sympathetic ophthalmia (or sympathetic ophthalmitis) is the much dreaded condition in which serious inflammation attacks the sound eye after injury of the other eye. In recent years sympathetic ophthalmia has become a rare disease, in spite of the fact that ophthalmic surgery has become more conservative. Though common in the American Civil and Franco-Prussian Wars, it was very rare during the two World Wars. This gratifying fact is due to increased skill in the treatment of perforating wounds, particularly in the application of antiseptic principles. A perforating wound, especially if a foreign body is retained within the eye, is, however, a source of great anxiety to the most experienced surgeon.

Sympathetic ophthalmia almost always results from a perforating wound, especially such as is caused by a foreign body which remains within the eye. Wounds in the ciliary region—the so-called "dangerous zone"—involving the ciliary body and leading to its incarceration in the scar, have always been considered specially dangerous; it is doubtful if, *per se*, they are more dangerous than others. On the other hand, it is certain that wounds in which iris, ciliary body or lens capsule is incarcerated are more likely to set up sympathetic ophthalmia than others. If suppuration supervenes sympathetic ophthalmia is very unlikely to follow; hence perforating ulcers very rarely cause it. It is also extremely rare without perforation, if indeed it ever occurs in these circumstances.

Children are particularly susceptible, but it occurs at any age. It usually begins four to eight weeks after the injury to the first eye (the exciting eye) has taken place, rarely earlier, but the onset may be delayed for many months or even years —it is said as many as 40 years.

There is always iridocyclitis in the exciting eye. Usually it is a plastic iridocyclitis which has been set up by injury and has not subsided in the course of three or four weeks. Instead of quieting down the ciliary injection remains, there is lacrymation and the eye is tender: special attention should be

directed to the presence or absence of precipitates ("k.p.") on the back of the cornea. In the rarer cases of delayed sympathetic ophthalmia the exciting eye has passed into a quiescent state. It may have shrunken completely. The onset of sympathetic ophthalmia in the second eye is then often ushered in by return of irritation—ciliary injection, tenderness, &c.—in the shrunken globe. The exciting eye, while showing evident traces of old iridocyclitis, may yet possess useful vision.

Sympathetic ophthalmia—the disease in the second or sympathising eye—is almost always a plastic iridocyclitis differing in no respect from this form of iridocyclitis due to other causes. In rare cases it manifests itself as a neuro-retinitis or choroiditis. In cases which the surgeon knows to be liable to the condition the first sign may be the presence of precipitates ("k.p.") on the back of the cornea, noticed at this early stage because they have been dreaded and carefully watched for. In other cases the patient first seeks advice for defective vision or inflammation in the uninjured eye (sympathetic irritation).

Prodromal symptoms are sensitiveness to light and transient indistinctness of objects. The latter is due to weakness of accommodation; objects become blurred when doing fine work, but after an interval of rest vision improves. On examination at this stage there may be lacrymation, slight ciliary injection, tenderness of the eyeball, as shown by the patient shrinking from an attempt at examination, precipitates on the back of the cornea, and vitreous opacities. The prodromal symptoms may occur in intermittent attacks, spread over a considerable period.

When fully developed all the signs and symptoms of iridocyclitis (*q.v.*) are present, varying in degree according to the severity of the case. The prognosis as to vision is always doubtful, but if there is much deposition of plastic exudates in the pupillary area it becomes extremely grave. Cases showing little exudation ("serous iritis"), but a deep anterior chamber and "k.p.," have a more favourable prognosis, but they may at any moment develop into the severe plastic type. Tension, difficult to determine on account of tenderness, is moderately raised in the early stages. It may then pass into the condition of lowered tension with gradual shrinking of the globe; or the iridocyclitis may subside, the eye quieting down and retaining fair vision. In the worst cases a ring synechia forms and secondary glaucoma supervenes (*vide* p. 282), or both occlusio and seclusio pupillæ or total posterior synechia

(*vide* p. 260) occur and the eye shrinks. Sympathetic ophthalmia sometimes takes two or more years to run its course.

The *pathology* of sympathetic ophthalmia is unknown. The microscopic features in both the exciting and the sympathising eye are the same.. In the earliest stages examined there are nodular aggregations of small round cells scattered throughout the uveal tract. In later stages the infiltration becomes diffuse, and epithelioid and giant cells appear; in fact, the condition is scarcely distinguishable from tubercle of the uveal tract. These are merely the signs of reaction to a constant, relatively mild form of irritation, and the view that the disease is tuberculous (Meller) is improbable. The ordinary signs of uveitis and its consequences are present.

The evidence which has accumulated in modern times tends to show that sympathetic ophthalmia is an infective disease. It is least liable to occur in otherwise likely cases if the wound or the retained foreign body is sterile. On the other hand, it very rarely occurs if actual suppuration has taken place in the exciting eye; possibly this may be due to some specific organism being destroyed by the superabundant growth of pyogenic organisms. It is more likely to occur from retention of shot, a chip of stone, glass, china, &c., than from that of a particle of hot steel, probably because the latter is sterile.

Sterility of an ocular wound is usually judged by a satisfactory course of healing and the absence of suppuration. It is by no means certain that all such wounds are, strictly speaking, sterile. The resistance of the patient's tissues has to be taken into account. With the same precautions a cataract wound may heal readily in a healthy man, but only after prolonged subacute iridocyclitis in a weakly patient. In many of these cases there are reasons for delayed cicatrisation, such as incarceration of the iris, synechia of lens capsule, &c., but the exact mode in which they act is a matter of conjecture.

Various theories have been brought forward to explain the occurrence of inflammation in the sympathising eye. It has been suggested that severe inflammation in one eye produces a tendency to ciliary irritation in the other eye by some occult means connected with their anatomical and physiological symmetry; there is no evidence to support this conjecture. More probable, *à priori*, is the view that infection travels along the optic nerve *viâ* the chiasma. On this theory one would anticipate neuro-retinitis in the sympathising eye as the most frequent manifestation of the disease, but it is extremely rare. The experiments supporting this theory fail to substantiate it.

A more probable theory is that there is a specific organism or virus, which has as yet escaped observation, possibly because it is ultra-visible by the microscope, but one which causes general infection through the blood stream. It may be that in addition to the infection there is an allergic factor, since it has been shown that uveal pigment can act as an antigen. It may be conjectured that the organism is harmless to other organs of the body, and that it finds a suitable nidus only in the other eye, perhaps owing to allergic hypersensitivity of the uveal tract. This theory explains best the facts of both ordinary and anomalous cases. When sympathetic ophthalmia supervenes after the injured eye has long been shrunken it may be conjectured that the organism has lain quiescent and encapsuled. The fact that in such cases the shrunken eye is again injured or becomes spontaneously irritable and inflamed shortly before the outbreak of inflammation in the other eye lends colour to this view. Cases in which the injured eye is excised and sympathetic ophthalmia is said to supervene many years afterwards are best explained as ordinary iridocyclitis—a by no means rare disease—occurring quite independently of the injury.

The *treatment* of sympathetic ophthalmia is one of the most difficult problems in ophthalmology, and often demands the exercise of great judgment.

It is, in the first place, prophylactic. In every case of perforating wound, with or without the retention of a foreign body, the question of excision of the eye on account of danger to its fellow arises. It may be assumed as an axiom that *sympathetic ophthalmia never occurs after the excision of an injured eye unless it has already commenced at the time of operation.* Hence, early excision is a positive safeguard against the disease. The injury to the eye may, however, be otherwise trivial, so that restoration of good sight may be possible. The rule should be to excise any eye which is so injured that it is improbable that useful vision will be regained. In cases where this is doubtful expectant treatment may be adopted for a time. If the eye quiets down quickly it is unlikely to set up sympathetic. What, then, are the chief causes which keep up irritation? The most important are entanglement of the iris or ciliary body or lens capsule in the wound, and the presence of a retained foreign body. Every effort must therefore be made to free the iris or ciliary body from the wound by excision of any prolapse, followed, if necessary, by division of anterior synechiæ. Upon the success of these efforts the retention of

(*vide* p. 260) occur and the eye shrinks. Sympathetic ophthalmia sometimes takes two or more years to run its course.

The *pathology* of sympathetic ophthalmia is unknown. The microscopic features in both the exciting and the sympathising eye are the same. In the earliest stages examined there are nodular aggregations of small round cells scattered throughout the uveal tract. In later stages the infiltration becomes diffuse, and epithelioid and giant cells appear; in fact, the condition is scarcely distinguishable from tubercle of the uveal tract. These are merely the signs of reaction to a constant, relatively mild form of irritation, and the view that the disease is tuberculous (Meller) is improbable. The ordinary signs of uveitis and its consequences are present.

The evidence which has accumulated in modern times tends to show that sympathetic ophthalmia is an infective disease. It is least liable to occur in otherwise likely cases if the wound or the retained foreign body is sterile. On the other hand, it very rarely occurs if actual suppuration has taken place in the exciting eye; possibly this may be due to some specific organism being destroyed by the superabundant growth of pyogenic organisms. It is more likely to occur from retention of shot, a chip of stone, glass, china, &c., than from that of a particle of hot steel, probably because the latter is sterile.

Sterility of an ocular wound is usually judged by a satisfactory course of healing and the absence of suppuration. It is by no means certain that all such wounds are, strictly speaking, sterile. The resistance of the patient's tissues has to be taken into account. With the same precautions a cataract wound may heal readily in a healthy man, but only after prolonged subacute iridocyclitis in a weakly patient. In many of these cases there are reasons for delayed cicatrisation, such as incarceration of the iris, synechia of lens capsule, &c., but the exact mode in which they act is a matter of conjecture.

Various theories have been brought forward to explain the occurrence of inflammation in the sympathising eye. It has been suggested that severe inflammation in one eye produces a tendency to ciliary irritation in the other eye by some occult means connected with their anatomical and physiological symmetry; there is no evidence to support this conjecture. More probable, *à priori*, is the view that infection travels along the optic nerve *viâ* the chiasma. On this theory one would anticipate neuro-retinitis in the sympathising eye as the most frequent manifestation of the disease, but it is extremely rare. The experiments supporting this theory fail to substantiate it.

A more probable theory is that there is a specific organism or virus, which has as yet escaped observation, possibly because it is ultra-visible by the microscope, but one which causes general infection through the blood stream. It may be that in addition to the infection there is an allergic factor, since it has been shown that uveal pigment can act as an antigen. It may be conjectured that the organism is harmless to other organs of the body, and that it finds a suitable nidus only in the other eye, perhaps owing to allergic hypersensitivity of the uveal tract. This theory explains best the facts of both ordinary and anomalous cases. When sympathetic ophthalmia supervenes after the injured eye has long been shrunken it may be conjectured that the organism has lain quiescent and encapsuled. The fact that in such cases the shrunken eye is again injured or becomes spontaneously irritable and inflamed shortly before the outbreak of inflammation in the other eye lends colour to this view. Cases in which the injured eye is excised and sympathetic ophthalmia is said to supervene many years afterwards are best explained as ordinary iridocyclitis—a by no means rare disease—occurring quite independently of the injury.

The *treatment* of sympathetic ophthalmia is one of the most difficult problems in ophthalmology, and often demands the exercise of great judgment.

It is, in the first place, prophylactic. In every case of perforating wound, with or without the retention of a foreign body, the question of excision of the eye on account of danger to its fellow arises. It may be assumed as an axiom that *sympathetic ophthalmia never occurs after the excision of an injured eye unless it has already commenced at the time of operation.* Hence, early excision is a positive safeguard against the disease. The injury to the eye may, however, be otherwise trivial, so that restoration of good sight may be possible. The rule should be to excise any eye which is so injured that it is improbable that useful vision will be regained. In cases where this is doubtful expectant treatment may be adopted for a time. If the eye quiets down quickly it is unlikely to set up sympathetic. What, then, are the chief causes which keep up irritation? The most important are entanglement of the iris or ciliary body or lens capsule in the wound, and the presence of a retained foreign body. Every effort must therefore be made to free the iris or ciliary body from the wound by excision of any prolapse, followed, if necessary, by division of anterior synechiæ. Upon the success of these efforts the retention of

the eye may depend. If they fail, which is more likely to be the case if the lens is also wounded, ciliary injection is certain to continue.

During this expectant period the most careful watch is kept for "k.p." If the eye continues irritable, with ciliary injection, photophobia, and lacrymation, and above all if "k.p." appears, the eye should be excised. It is seldom wise to wait longer than a fortnight unless there are undoubted signs of amelioration. The slightest sign of ciliary irritation or "k.p." in the other eye indicates the necessity for immediate excision of the injured eye. It must be remembered that children are more susceptible than adults. Care must be taken not to confuse a simple conjunctivitis with ciliary irritation.

Even more difficult to decide is the treatment in those cases in which sympathetic ophthalmia has already supervened. If the case is seen early, shortly after the onset of inflammation in the sympathising eye, and if the injured or exciting eye has no useful vision, this useless eye should be excised at once. There is no question that the excision of the exciting eye has a good effect upon the process in the sympathising eye if performed early. At a later stage there is no evidence to show that it exerts any influence at all.

The chief difficulty arises when the exciting eye has useful vision and the inflammation in the sympathising eye is severe. If this is the condition soon after the injury it may be wise to excise the injured eye. If, however, a considerable time has elapsed since the injury, excision of the exciting eye is likely to have little or no influence upon the process. Moreover, in the end the injured eye may have better vision than the sympathising one, for if the iridocyclitis is severe the sympathising eye may be lost in spite of all efforts. Under these conditions, therefore, the injured eye should be retained.

The treatment of the sympathetic iridocyclitis is that of iridocyclitis in general (*vide* p. 275). In addition to atropine, hot bathings, rest in a dark room, leeches, &c., the patient should be brought rapidly and thoroughly under the influence of mercury. Mercurial inunctions should be pushed, so that salivation occurs within a week, and the patient should then be kept on the border line of mercurialisation for a considerable period. Massive doses of sodium salicylate have proved beneficial in some cases. In later stages pilocarpine injections and the administration of iodides may assist in the absorption of exudates, and lead to improvement of vision. Perseverance

in these measures, aided by general tonic treatment, is of the utmost importance.

Improvement of vision may occasionally be obtained by operation, but no such interference is to be contemplated until all inflammation has subsided and the eye has been quiet for several months. In the milder cases an optical iridectomy may do good. In the worst cases, so long as there is perception, and moderately good projection of light, more desperate operations, such as extraction of the lens, &c., may be justifiably undertaken if the other eye is blind or has been removed.

CHAPTER XXII

Operations upon the Eyeball

Bacteriological Precautions

Before intra-ocular operations, particularly major operations such as cataract extraction, a bacteriological examination of the conjunctival sac should be made and, at any rate temporary surgical cleanliness attained by penicillin (*vide* p. 696); Dacryocystitis should be looked for and, if necessary, dealt with (p. 653). Ideally a culture should be taken and the measures already described as a preliminary to a cataract operation be adopted (*vide* p. 314).

Anæsthesia for Eye Operations

In most ophthalmic operations, and particularly in intra-ocular operations, it is desirable to have the conscious co-operation of the patient and to avoid the risks of the after effects of general anæsthesia. Hence local anæsthesia, with or without pre-medication, should be used. While the co-operation of the patient is always desirable, it is not always given, and it is unfair in the trying circumstances of an operation to expect that it should be. It is easy by adequate anæsthesia and akinesia to ensure that events will not get out of hand. In the case of neurotic and highly strung patients the previous administration of a sedative may suffice; if not, a general anæsthetic must be used.

Local Anæsthesia. (1) *Surface Anæsthesia.* Cocaine and its many derivatives are readily absorbed by the conjunctiva and cornea, and produce complete anæsthesia after instilling 4 drops of a 2 per cent. solution at intervals of five minutes. The iris, however, is not rendered completely anæsthetic by this method. Cocaine occasionally causes alarming symptoms in old and debilitated patients and as an idiosyncrasy, but this is very rare: as a precaution sustained pressure over the lacrymal sac during instillation will prevent absorption from the nasal mucous membrane. Pantocain is preferable to cocaine as it neither damages the corneal epithelium nor does it dilate the pupil. The cornea remains clear, and the sense

of pressure is abolished, so that the weight of instruments is not felt. Pantocain (also known as dessicain) is a novocain derivative, readily soluble in water, is stable when boiled, and mixes well with adrenaline. It produces a burning sensation and blepharospasm for about a minute, and a slight hyperæmia which disappears in three to five minutes.

(2) *Infiltration and Regional Anæsthesia.* Infiltration anæsthesia with novocain and adrenaline (novocain 2 per cent., adrenaline 1 in 10,000) or a suitable substitute is employed for operations on the lids for the removal of growths, injuries and plastic repairs, and electrolysis of the lashes. The lid margin is difficult to anæsthetise; a fine needle should be used and the injection given slowly and thoroughly. To effect anæsthesia of the iris 1 c.c. of novocain (4 per cent.) is injected into Tenon's capsule about half-way between the temporal border of the superior rectus and the upper edge of the external rectus. A fine needle is used, and care is taken to insert it very obliquely through the conjunctiva, to avoid the site of a vena vorticosa, and to keep close to the sclera. The injection is made just behind the equator. This is safer than giving a retro-ocular injection of 1 c.c. of novocain (4 per cent.), a procedure in which a fine needle, 5 cm. long, is passed through the skin of the lower lid along the outer wall of the orbit for 4 cm. and then turned medially for about 1 cm.

In cases when a general anæsthetic is undesirable the eye can be removed painlessly by an injection of 6 c.c. of novocain and adrenaline into the apex of the orbit.

Pre-medication. Morphia, heroin, omnopon, and scopolamine are commonly used before operation to allay fear and facilitate the quiet induction of general anæsthesia. They are liable to cause post-anæsthetic vomiting and constipation, and should only be used when specially indicated or as a preliminary in basal narcosis. Luminal, gr. i, taken by the mouth one hour before operation, is useful in rendering most adults quiet, yet still co-operative. Nembutal is usually more effective. A capsule containing 1½ grs. is given the night before operation, and one or two capsules, according to the patient's weight and age, one hour before operation. Its action, however, varies considerably, being without effect in some persons or causing a restless semi-consciousness in others.

General Anæsthesia. *Intravenous Administration of Drugs.* The best drug for this purpose is pentothal sodium, a rapidly acting barbiturate which produces a fall of blood pressure and intraocular pressure, good relaxation and moderately

quick recovery of consciousness. A single injection suffices for an operation of ten to twenty minutes' duration. For longer operations more of the solution is injected periodically. Conjunctival and corneal reflexes are abolished, but it is wise to use pantocain drops, injection of novocain and adrenaline into Tenon's capsule, and facial nerve block. During anæsthesia the eyes are central and directed slightly upwards.

Inhalation anæsthesia is indicated in operations upon children for squint, application of radon seeds to the sclera, &c., for prolonged plastic operations, operations upon the orbit, and excision of the eye. In all other ophthalmic operations it is preferable to use local anæsthesia with or without basal narcosis.

The chief objections to inhalation anæsthesia for intraocular operations are the deviation of the eyes, vascular congestion, and post-operative restlessness and vomiting.

Akinesia for Eye Operations

In intra-ocular operations involving a wide opening of the globe (*e.g.*, cataract) temporary paralysis of the orbicularis muscle is advisable in order to prevent squeezing together of the lids. For this purpose 4 to 5 c.c. of novocain and adrenaline are injected down to the periosteum covering the neck of the mandible where the upper branches of the facial nerve pass forwards and upwards. The patient should be instructed to open his mouth, and the position of the condyle and temporomandibular joint is located by the operator's left forefinger. After closing the jaw a point ½ inch below the position of the condyle is selected for the insertion of the needle, which should pass straight down to the periosteum. Four or 5 c.c. are injected, and after withdrawing the needle firm pressure and local massage are applied. Paralysis of the orbicularis rapidly follows.

An alternative method is injection across the branches of the facial nerve as they traverse the malar bone. A fine needle, about 1¾ inches in length, is inserted down to the periosteum of the malar bone at a point about 1 cm. below and behind the outer canthus. The needle is passed upwards towards the temporal fossa, forwards and downwards towards the infraorbital foramen, and downwards and backwards towards the tragus for an inch or so (Fig. 239).

The frontalis muscle and the supraorbital nerve may also be infiltrated by inserting the needle 1 cm. above the external angular process and passing it close to the periosteum along

the supraorbital margin until the supraorbital notch is reached. The advantage of this method is that it provides regional anæsthesia as well as paralysis of the orbicularis muscle.

When the lids have been paralysed in this way, in order to ensure post-operative closure a stitch should be inserted into the skin of the upper lid 3 mm. above its margin. This is fixed to the cheek by adhesive plaster after the operation and removed at the first dressing.

Fixation of the globe by a stitch passed through the tendon of the superior rectus muscle is necessary in intra-ocular operations conducted under general anæsthesia and is advisable

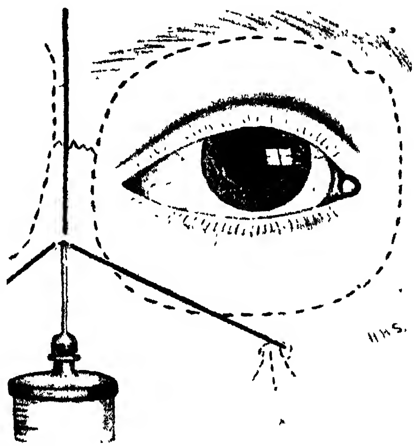


FIG. 239.

in those cases wherein the co-operation of the patient in looking down is in doubt. In fact, it is a safe precaution in all intra-ocular operations wherein looking down is essential. It is more effective after the injection of 0·5 c.c. novocain into the belly of the muscle. During the operation the stitch is either clamped without tension to the head towels by artery forceps or controlled by an assistant (Fig. 268).

The Speculum. In most operations a simple speculum (Lang's, Fig. 118) is adequate. When the eye is widely opened (as for cataract) a modification of Arruga's speculum is safer, whereby the weight is taken on the bridge of the nose and the temple, and the lids and speculum are lifted off the globe (Fig. 240). The simplest and safest method is to employ lid stitches. The upper

lid stitch (*vide supra*) instead of being inserted in the middle is passed through the skin twice at the junctions of the middle third with the outer and the inner thirds, and it, as well as the central superior rectus stitch, is clamped on the head towels to keep the upper lid back. A skin stitch in the lower lid depresses it by the weight of an artery forceps hanging down over the cheek.

General Routine. The preparation of the eye immediately before the operation is the same in all cases. The eyelashes are cut short and the eye is irrigated with normal saline solution. The skin of the lids is dried and painted with spirit or iodine. Two or three drops of penicillin (1,000 units per c.c.) are instilled. Five drops of pantocain (1 per cent.) are instilled at 3 minute intervals and then two drops of adrenaline (1 in 1,000). One drop of pantocain is also instilled into the

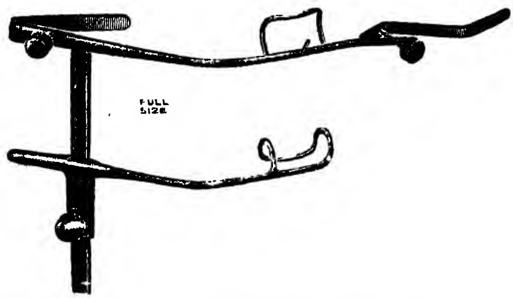


FIG. 240.—Arruga-Stallard speculum.

other eye to diminish the risk of closure of the lids. The orbicularis oculi is paralysed (*vide supra*); an injection of novocain is made into Tenon's capsule if necessary (*vide supra*). Sterile towels are draped round the head, neck and chest and the face covered with a gauze mask in which an aperture is cut to give access to the eye. The lid stitching is inserted after a minute local injection of novocain into the skin (*vide supra*). The speculum (if employed) is inserted and the eye is ready for operation.

OPERATIONS UPON THE CORNEA

Paracentesis for hypopyon ulcer has already been described (*vide* p. 209). Paracentesis for cyclitis is performed in identically the same manner as in the second method there described.

Operations upon the Iris

Iridectomy, which consists in the excision of a portion of the iris, is performed for the following conditions:—(1) Prolapsed iris; (2) corneal or lenticular opacities (optical iridectomy); (3) glaucoma; (4) as a preliminary or as part of cataract extraction; (5) threatening ring synechia (*vide* p. 271); (6) ectatic corneal cicatrices; (7) foreign bodies in, or small cysts or tumours of the iris.

Iridectomy for prolapsed iris has already been described (*vide* p. 212).

Optical iridectomy is indicated in some cases of localised opacities of the cornea or lens, very rarely for occlusion of the pupil or subluxation of the lens. In all cases there must be proof or good reason to believe that the light percipient structures are capable of performing their functions. If the patient is old enough to have the vision tested this should show improvement when the pupil is dilated by a mydriatic. The opacities must be localised, and in the case of lenticular opacities there must be good reason to assume that they are stationary. The results are frequently disappointing.

An optical iridectomy should be as narrow as possible, in order to avoid dazzling and to obtain an approximation to stenopæic vision (vision through a narrow slit, *vide* p. 23). It should not extend to the ciliary border. The site of election is down and in (Fig. 241), but in the case of corneal opacities the clearest region of the cornea must be chosen, unless this happens to be above, in which case the coloboma would be covered by the lid and useless for vision.

Instruments required: speculum, two pairs of fixation forceps, bent keratome, iris forceps or iris hook, de Wecker's scissors, iris repositor. General anæsthesia is only necessary in very young or neurotic patients.

The keratome is inserted at or just inside the apparent corneo-scleral margin, the blade being kept parallel to the plane of the iris. It is pushed on until the incision is sufficiently long. The handle is then depressed, so that the blade lies against the back of the cornea; the danger of pricking the lens with the point is thus reduced to a minimum. The keratome is then slowly withdrawn. The iris forceps are inserted closed, then opened very slightly and the iris seized just outside the pupillary margin: or the iris may be drawn out with a blunt iris hook, which allows a narrower coloboma to

be made. The iris is drawn out of the wound and a portion excised with de Wecker's scissors. A slit-like coloboma is made by holding the scissors so that the blades are in the direction of a radius of the iris (Fig. 243); the narrowest coloboma may be made by a simple radial iridotomy, no iris being removed. The iris is freed from the wound, unless already free, by the repositor. Sterile atropine ointment is introduced into the conjunctival sac, and the eye is bandaged.

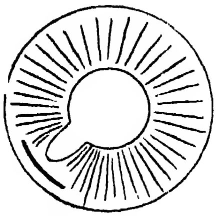


FIG 241.—Diagram of wound and coloboma in optical iridectomy at the site of election.

Iridectomy for Iritis (*vide* p. 271) should be performed as in optical iridectomy, but in the upper part of the iris.

Preliminary Iridectomy (*vide* p. 316). When iridectomy is done as a preliminary to cataract extraction it should be done with a keratome in the upper part of the cornea and in the same manner as an optical iridectomy. If there is raised tension (*vide* p. 316) the section should be large, and the iris should be torn away as in iridectomy for glaucoma (*vide infra*).

Iridectomy for glaucoma has for its object the opening up of a sufficiency of the angle of the anterior chamber to permit of efficient filtration of aqueous. It is essential, therefore, that the coloboma shall extend to the ciliary attachment of the iris, and that it shall be broad at the periphery. It has already been pointed out that when the iris is torn away the fracture occurs at the thinnest part, viz., at the ciliary attachment. This will generally happen in iridectomy for acute glaucoma, if the attack is the first or early in the history of the disease. In chronic glaucoma, however—and the same applies to an acute exacerbation occurring in the course of chronic or subacute glaucoma—the periphery of the iris is firmly adherent to the corneosclera. When the iris is torn away the fracture will be at the false angle, and filtration of lymph will not be facilitated. The rules usually given as guidance to the correct performance of iridectomy for glaucoma are that the section shall be peripheral and the coloboma wide. From the above remarks it will be seen that a very peripheral section is not very important in true acute glaucoma, but that it has usually been thought of importance in chronic glaucoma.

In acute glaucoma and in acute exacerbations of the chronic form a general anæsthetic or deep regional anæsthesia into

Tenon's capsule is required, chiefly because the high tension prevents sufficient absorption of cocaine to render the cornea —much less the iris—anæsthetic, partly also because the patient's self-control has been shattered by pain and anxiety. Control of the eye by a superior rectus stitch is also advisable. Eserine should always be instilled into the unaffected eye (*vide* p. 292).

In cases of acute glaucoma with very shallow anterior chamber, a preliminary posterior sclerotomy by diathermy or a Graefe knife (*vide* p. 481) may be done ten minutes or so before the iridectomy; but this is seldom necessary if the treatment suggested on p. 292 is carried out.

Instruments required: speculum (Fig. 240), two pairs of fixation forceps, narrow Graefe cataract knife, iris forceps, de Wecker's scissors, iris repositor.

The surgeon stands above the patient, using his right hand to make the section for the right eye, the left hand for the left eye. The knife is held with its plane parallel to the plane of the iris, care being taken that the back of the knife is away from the surgeon. (It is an extremely awkward accident to introduce the knife with the back upwards.) The point is introduced at least 2 mm. behind the apparent corneo-scleral margin. It is inserted at the point corresponding with seven minutes to (right eye) or past (left eye) twelve on a clock face (Fig. 242). As soon as the point is in the anterior chamber it will look much brighter than the part in the corneo-sclera; if this is not noticeable it is probable that the knife is badly directed and is burrowing in the cornea. It is passed steadily onwards across the anterior chamber to a spot corresponding with seven minutes past or to twelve on the dial of a clock, where the counter-puncture is made. In glaucoma, especially acute glaucoma, the anterior chamber is very shallow, so that it may be very difficult to pass the knife across without catching in the iris and wounding the lens, a most disastrous accident. The deepest part of the chamber is at the periphery, and it will usually be found easiest to coax the point of the knife round the periphery, gently pushing the iris away with the back.

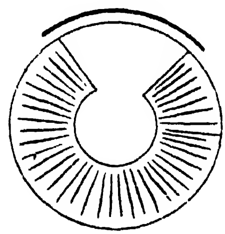


FIG. 242.—Diagram of wound and coloboma in glaucoma iridectomy.

As soon as the counter-puncture is made the knife is pushed

on until 5 or 6 mm. are exposed. Cutting out is performed by a series of small sawing movements, little pressure upwards being required with the very sharp knife. It is very necessary to use these sawing movements properly, as the sharpest knife fails to cut if it is simply pressed hard against a surface.

In this manner the section through the corneo-sclera is kept at a uniform distance of 1 or 2 mm. behind the apparent corneo-scleral margin. Some conjunctival flap has already been cut at the sides, but the middle of the knife blade is still under the conjunctiva. The edge of the knife is then directed forwards

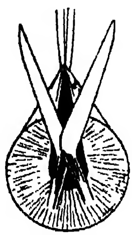


FIG. 243.

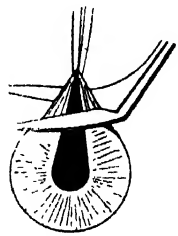


FIG. 244.

FIG. 243, iridectomy with blades of de Wecker's scissors held radially; FIG. 244, with blades held at right angles to the previous position. The former method results in a narrower coloboma, such as is preferable in optical iridectomy and in extraction of senile cataract with iridectomy.

and the conjunctiva cut through by one or two sawing movements.

During these manœuvres care must be taken that the points of the fixation forceps are not pressing into the globe, which is very likely to occur owing to the attention being concentrated upon the section.

While the eye is gently rotated downwards by the superior rectus stitch, the surgeon takes the iris forceps in his left hand and de Wecker's scissors in his right. With a sterile swab the assistant turns down the conjunctival flap so that it lies over the cornea, unless this has been done previously with the back of the knife after completion of the section. The points of the closed forceps are then inserted in the wound and carried to a point half-way between the pupillary and ciliary margins of the iris at a point slightly to the right of the vertical meridian of the cornea. The forceps are opened slightly, and the iris gripped. The iris is pulled out and cut to the right side of the forceps. This stroke makes the right

limb of the coloboma. The iris is then drawn across towards the left. By this movement it is torn from its attachment for the whole width of the section. It is then drawn a little back towards the right, so that it may not be jammed into the left angle of the incision. The free part is then cut off by a second snip of the scissors. This stroke makes the left limb of the coloboma.

The iris repositor is then taken by the surgeon. The tip of the repositor is introduced into the wound and insinuated between the cornea and the iris on one side. By a radially directed movement the iris is smoothed out towards the centre of the pupil so that if the edge of the coloboma is caught in the angle of the wound it will be freed. The same manœuvre is repeated on the other side of the wound. This part of the operation is very important, and may be very difficult. Only when the edge of the pupil is in its natural position and looks circular but for the small defect in the upper part is the surgeon convinced that the pillars of the coloboma are free from entanglement in the wound. The conjunctival flap is then turned back over the wound with the iris repositor, care being taken that it is not folded upon itself. Any blood clot is carefully removed with forceps or a swab. The surgeon lifts the speculum away from the eye, at the same time seizing the lid suture. Removing the speculum, he lifts the upper lid over the wound in such a manner as to prevent the lid from displacing the conjunctival flap.

FIG. 245.—Drawing up of the iris after extraction of cataract with incarceration of the pillars of the coloboma.

Both eyes are bandaged. The unoperated eye may be uncovered after two days.

The chief complications which may arise during the operation are hæmorrhage into the anterior chamber (not usually serious, but inconvenient); wound of the lens (often not discovered until opacity develops); severe intraocular hæmorrhage, leading sometimes to extrusion of the lens, vitreous, and even retina.

It may be mentioned that some surgeons use a keratome in this operation. Some also cut off the iris with one snip of the scissors, the blades being directed at right angles to the direction of the forceps (Fig. 244). It is difficult to imagine how this can produce an absolutely peripheral coloboma.

The after-treatment consists in complete rest in bed. The eye is dressed once daily; neither mydriatic nor miotic is instilled unless complications supervene.

The chief complications arising after the operation are extrusion of the lens (due to too large a section), injury to the eye by the patient (usually during sleep) (*vide* p. 498), severe intraocular hæmorrhage, &c. Delay in re-formation of the anterior chamber for several days may happen; though undesirable, it will probably lead to no ill effects. The wound may bulge, with or without prolapse of iris or incarceration of the angles of the coloboma in the wound. A cystoid cicatrix may result, not altogether undesirable from the point of view of filtration in chronic glaucoma, but liable to arouse iridocyclitis or permit infection and panophthalmitis. Bulging of the wound may be due to partial subluxation of the lens, which may necessitate extraction under grave technical difficulties. Wound of the lens during the operation leads to traumatic cataract, also demanding extraction.

Iridotomy is section of the iris without excision of any portion. It is employed for making a new pupil when the normal pupil is closed or has been drawn up to the wound of a faulty cataract extraction with incarceration of the pillars of the coloboma (Fig. 245). In such a case it is usually done as follows. A keratome incision 3 or 4 mm. long is made near the periphery of the cornea at the most suitable part, usually the temporal side. The direction of the section should correspond with the position of the proposed puncture in the iris, *i.e.*, it will be approximately radial. This facilitates the opening and shutting of the iris scissors, and minimises the bruising of the lips of the wound. The closed blades, one of which is pointed, of de Wecker's scissors are passed into the anterior chamber. The pointed blade is forced through the iris and passed on horizontally. The blades are then closed, a horizontal slit being made in the iris. This cuts across the stretched fibres, which retract, leaving an oval artificial pupil.

Iridotomy may also be performed by sawing movements with Ziegler's sickle-knife (Fig. 259).

Sometimes the iris can be hooked out through a keratome incision by means of an iris hook, and a piece cut off. This gives a good pupil, but is of course strictly speaking an iridectomy.

The results of iridotomy for artificial pupil are often disappointing, the inflammatory reaction causing the gap to fill with exudate which organises into scar tissue. It is,

however, remarkable how little reaction follows in some cases, especially cases of syphilitic origin.

Iridotomy may be a necessary preliminary to iridectomy in cases of bombé iris. It is then usually done by passing a Graefe knife across the anterior chamber, puncturing and counter-puncturing both cornea and iris. The iridectomy is done before the punctures become closed with exudate, which usually occurs soon.

Division of Anterior Synechiæ is a form of iridotomy. The operation is too technical to be described in detail here.

Operations upon the Sclerotic

Sclerotomy. Puncture of the globe behind the equator (*Posterior Sclerotomy*) is sometimes performed by diathermy or a Graefe knife to reduce intraocular pressure temporarily in acute glaucoma.

Sclerectomy. *Anterior Sclerectomy* is the name given to various operations for chronic glaucoma in which a fragment of

FIG. 246.—Bowman's discission needle, with stop.

FIG. 247.—Saunders's discission needle, with long cutting

FIG. 248.—Lang's knife.

the sclerotic is excised. Removal by scissors, as in Lagrange's operation, is now almost entirely replaced by trephining.

Trephining was first used for glaucoma by Argyll Robertson (1876), and re-introduced in 1909 by Freeland Fergus and R. H. Elliot. The details of the modern method have been elaborated chiefly by Elliot. The operation is indicated in chronic glaucoma and infantile glaucoma, and has been re-

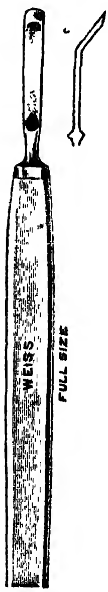


FIG. 249.—
Tooke's angled
corneal splitter.

FIG. 250 —
Trephine.

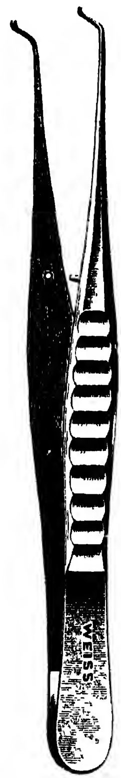


FIG. 251.—
Elliot's disc forceps.

commended in some forms of secondary glaucoma, *e.g.*, that following cataract extraction, anterior staphyloma, &c.

Instruments required: Lang's speculum (Fig. 118), two pairs of fixation forceps (ordinary and claw), tenotomy scissors, iris repositor, Lang's knife (Fig. 248), or Tooke's angled corneal splitter (Fig. 249), 1·5 mm., trephine (Fig. 250), straight iris forceps or Elliot's disc forceps (Fig. 251),

de Wecker's scissors, needles, and needle-holder. The eye having been prepared and anæsthetised as indicated (p. 474), the conjunctiva is seized with ordinary fixation forceps 8 or 9 mm. above the cornea. A large conjunctival flap is made, almost concentric with the margin of the cornea (Fig. 252) : the lower ends of the wound should be well away from the limbus, otherwise filtration is likely to be impeded owing to cicatricial tissue. The flap is dissected down to the upper part of the corneal margin and turned down over the cornea. It is kept stretched in this position with the iris repositor laid horizontally upon it. The subconjunctival tissue is divided with the Lang's knife or Tooke's angled splitter, the utmost

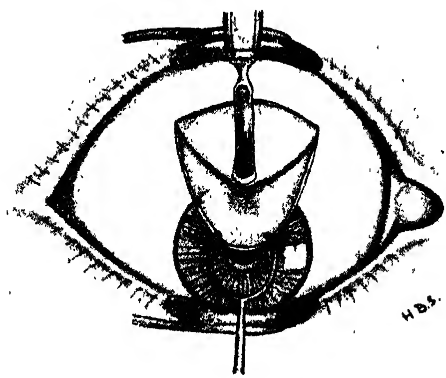


FIG. 252.

care being taken to avoid button-holing the flap. The edge of the cornea is thus clearly defined, and the dissection is carried into the cornea so that the superficial lamellæ are dissected up with the flap for about 1 mm. The trephine is then applied, so that half the aperture lies on the cornea, the other half on the sclera. The corneo-sclera disc is cut by a few rotatory movements. When the anterior chamber is entered aqueous escapes, and the pupil is displaced upwards. The trephine is removed, a knuckle of iris protrudes from the wound and the disc is forced out. It usually remains attached by a small hinge. By tilting the trephine slightly forwards, so that the corneal side of the disc is cut rather more deeply than the scleral, it is generally possible to insure that the

hinge shall be on the scleral side. The disc is seized with Elliot's disc forceps and excised with de Wecker's scissors (Fig. 253). The root of the iris is then picked up with straight iris forceps, drawn slightly downwards and from side to side so as to produce a peripheral iridectomy. The cornea is gently stroked downwards with a repositor until the pupil is round and clear of the trephine hole. During these manœuvres the assistant must keep the flap well stretched downwards with the repositor (*not* forceps) so as to avoid it also being button-holed—a rather serious accident. The flap is then replaced, fixed at the summit by a single suture, and smoothed

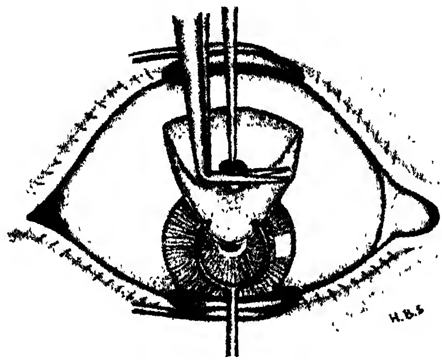


Fig. 253.

down; the speculum is removed, and the lid lifted over the wound.

There is a great tendency for iritis to develop immediately after trephining. Hence atropine should always be used on the following day and for several days afterwards.

The chief complications at the operation are making the trephine hole too peripheral, often with consequent escape of vitreous and incarceration of the ciliary processes; button-holing the flap either with the trephine or de Wecker's scissors; escape of the corneo-scleral disc into the anterior chamber; and non-presentation of iris in the wound. The last complication is usually due to slow escape of aqueous owing to the trephine being blunt.

There is often considerable delay in the re-formation of the anterior chamber. In rare cases it never re-forms, the lens becomes opaque, and vision is usually lost.

Later complications are iritis, detachment of the choroid (*vide* p. 348), blockage of the wound with iris, ciliary body, lens or vitreous, failure of filtration from dense cicatrisation, &c. Owing to the prominence and thinness of the overlying conjunctiva late infection may occur long after the operation—it occurred in only 14 cases out of 536 trephinings at Moorfields Eye Hospital (Davenport).

Operations upon the Lens

Discission or **Needling** of the intact lens should rarely be performed after fifteen years of age; it may be employed up to thirty or even thirty-five, but the nucleus of the lens is then likely to give trouble. It is indicated in most cases of dense lamellar cataract, some cases of congenital cataract, and some cases of high myopia. Discission is used at any age for the division of dense secondary cataract (after-cataract).

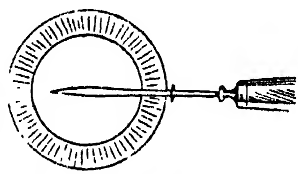


Fig. 254.—Diagram of discission with one needle.

Needling of the soft lens in young patients usually requires a general anæsthetic, though it is quite painless under cocaine. The pupil must be fully dilated with atropine.

Instruments required: speculum (Fig. 240), fixation forceps, cataract needle (Figs. 246–247). I prefer a needle with a fairly long cutting edge. It is best to perform the operation in a darkened room with oblique illumination. The surgeon stands above the patient. The conjunctival sac having been douched, and the speculum inserted, the eye is fixed down and in (right eye) with fixation forceps held in the left hand. The needle is introduced just outside the limbus (Fig. 254), *i.e.*, through the conjunctiva and sclero-cornea, in a plane parallel to that of the iris, at a point just above the horizontal meridian of the cornea. It is carried through the anterior chamber until the point reaches the lower part of the pupil. The handle is then slightly raised, so that the point just perforates the lens capsule. The handle is then moved so that it and the point move through arcs of circles which have their centre at the spot where the shaft is engaged in the corneo-sclera. Having thus made a curved, more or less vertical incision in the capsule, a second incision is made at right angles to it. This is done by very slightly withdrawing the needle so as

to disengage it. It is then passed farther on towards the left side of the pupil. The handle is again slightly raised, and at the same time rotated, so that the cutting edge is brought in contact with the capsule. As the needle is slowly withdrawn a straight incision is made in it in a horizontal direction. When this is sufficiently large the handle is depressed. The handle is rotated so that the plane of the blade faces upwards, and the needle is quickly withdrawn from the eye. By withdrawing it quickly no aqueous should be lost. If much aqueous is lost, anterior synechia may result. Sterile atropine ointment is introduced into the conjunctival sac, and both eyes are bandaged.

FIG. 255.—Ziegler's knife.

The most important point about after-treatment is keeping the pupil well dilated, which is done by atropine ointment three or four times a day. There is always some ciliary reaction. The amount of swelling of the lens fibres depends upon the size of the incisions in the capsule, but also varies with different lenses. The reaction is often very slight in cases of true congenital cataract; in these cases the iris responds scarcely at all to atropine. If it is particularly desired to avoid the necessity of a subsequent curette evacuation the incisions should be quite small in the first operation. If it is intended to perform a curette evacuation (*vide* p. 488) the incisions may be as large as possible, and the needle may even be introduced moderately deeply into the lens and the fibres broken up. In these cases, and occasionally when it is not anticipated, there is great swelling of the lens; the anterior chamber becomes filled with flocculent masses; there is intense ciliary injection accompanied by raised tension and pain. The tension sometimes subsides in a few hours with leeching, but if not must be relieved at once, or the sight will be seriously damaged by secondary glaucoma. A curette evacuation is then done (*vide* p. 488). In cases in which no curette evacuation is performed a second and third needling will usually be necessary before a clear opening is obtained. Intervals of several weeks or even months may elapse until the absorption set up by the previous operation is complete. The final needling will be of the type of a discission for secondary cataract.

Since time is of little importance in the case of children

compared with safety to the eye, I prefer to avoid curette evacuation in these cases if possible. Simple needling causes little reaction, and septic infection is very rare if a subconjunctival puncture is made, as recommended above. Excessive swelling of the lens and curette evacuation cause much more disturbance, and the risks of sepsis are greater, for the swollen lens substance is an excellent culture medium.

Discission of Secondary Cataract (after-cataract) (*Syn.—Capsulotomy*) is performed in exactly the same manner as discission of the soft lens if the after-cataract is not too dense to be divided by a single needle. Some surgeons advise needling every case of senile extracapsular extraction as soon as the eye has quieted down, *i.e.*, in a fortnight or three weeks: the capsule is then soft and easily divided. If there has been

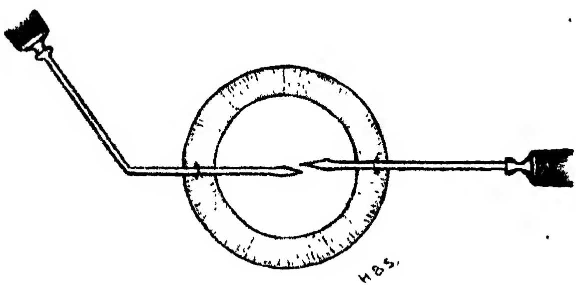


FIG. 256.—Diagram of discission with two needles.

iritis or iridocyclitis needling is contraindicated until all signs of inflammation have passed off; hence a careful search for "k.p." must be made in every case.

If the capsule is dense and thickened a Ziegler's knife may be used (Fig. 255), or two needles may be employed, the shaft of the one used on the nasal side being bent to an angle of 135°. By the latter method no undue strain is thrown upon the ciliary body, and the membrane is prevented from tearing away from the ciliary body instead of being torn in the centre. The eye is fixed by an assistant. The needles are introduced with the flat surfaces upwards through the cornea at opposite sides of it in the horizontal meridian, 3 or 4 mm. internal to the apparent margin (Fig. 256). They are passed through the centre of the membrane close to each other, and the cutting edges are swept in opposite directions, one up and the other down, care being taken that each needle rotates around the

spot on the shaft which is engaged in the cornea. In this manner the points are drawn apart, and the membrane is cut. A further attempt may be made to tear it in the direction at right angles. The needles are withdrawn quickly so that aqueous may not be lost. Atropine is instilled, and the eye is bandaged. There is seldom much reaction, unless the previous operation has been faulty; in such cases the additional irritation may precipitate an attack of sympathetic ophthalmia.

When the membrane is very dense it is often best to divide it with scissors, as in iridotomy (*vide* p. 480); or the very ingenious canula-scissors may be employed. They were invented many years ago, but fell into disrepute owing to the loss of eyes from sepsis. They should be kept in alcohol when not in use. A small peripheral puncture with a broad needle is first made, and through this the canula-scissors are introduced.

It may be mentioned that needling operations are by no means so simple as they appear in the hands of an experienced. operator. Every movement of the needle must be made round the spot where the shaft penetrates the globe.

Curette Evacuation or **Linear Extraction** is the operation whereby after discission, whether accidental (traumatic cataract) or intentional, the softened lens matter is let out of the anterior chamber.

Instruments required: speculum (Fig. 240), fixation forceps, bent keratome, curette (Fig. 258), lens expressor (Fig. 262), iris repositor, irrigation apparatus (*vide* p. 498), (toothed capsule forceps). Local anæsthesia suffices, except in the case of children or unruly patients.

The pupil must be fully dilated with atropine. The position of the section is of little importance; some surgeons place it above, where it is under the upper lid, others below. The surgeon stands accordingly either above or at the side of the patient.

The conjunctival sac having been douched and the speculum inserted, the eye is fixed with fixation forceps. The keratome is passed through the cornea, 1 mm. internal to the apparent margin (cf. wound in Fig. 241), with the blade parallel to the plane of the iris. It is pushed on until the incision is about 5 mm. long. The point may be dipped so as to pass into the lens without disadvantage, and the incision may be increased as the keratome is being slowly withdrawn by extending each angle, using the two edges of the keratome like knives. Toothed capsule forceps may be introduced into the anterior chamber,

opened for 3 mm., gently pressed into the capsule and closed. Movements of 2 mm. to the temporal and nasal sides and upwards will remove a piece of capsule, diminishing the risks of dense after-cataract in the pupillary area. The tip of the curette is then gently insinuated just within the edges of the wound, not quite into the anterior chamber. Slight pressure

FIG. 257. — Graefe cataract knife. A slightly broader knife is used in cataract extraction than in glaucoma iridectomy; it has the advantage of being less resilient. The knives used for glaucoma iridectomy are generally re-ground cataract knives.

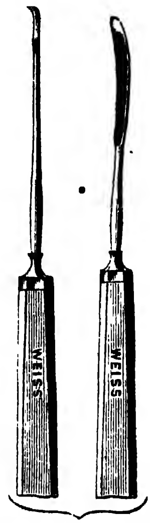


FIG. 258. — Cystotome (Moorfields pattern) and curette. (These should *not* be at the two ends of the same instrument.)

is exerted upon the peripheral lip, and the soft lens matter travels along the groove of the curette. No attempt should be made to remove the whole of the lens matter, on account of danger of rupture of the suspensory ligament and escape of vitreous; the remnants will be absorbed. There is no probability of the iris prolapsing or becoming incarcerated in the wound if it is properly dilated, but the repositor should be gently inserted so as to push back any lens capsule which

may have prolapsed. Sterilised atropine ointment is inserted, and both eyes are bandaged.

Usually the ciliary irritation is greatly benefited by the operation. The greatest care must be exercised as to surgical cleanliness, for most of the accidents from infection in treating soft cataract occur, not from needling, but from curette evacuation. Anterior synechia of iris or capsule, iridocyclitis, or sympathetic ophthalmia may occur, but are happily rare.

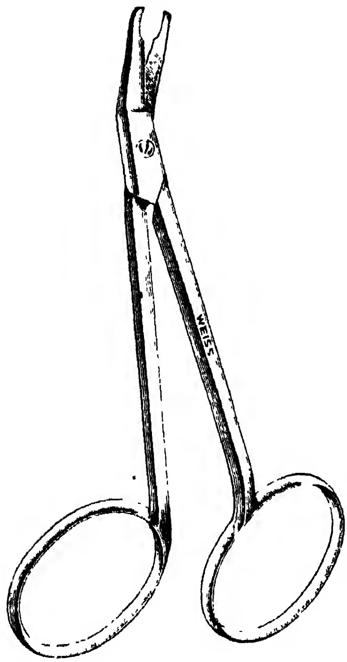


FIG. 259.—Sinclair's angular scissors.

Some surgeons extract soft cataracts by the linear method without previous discission. This mode of treatment is not advocated.

Extraction of Senile Cataract may be performed with or without iridectomy. The iridectomy is performed by some surgeons six weeks or more before extraction. I advise this method only in certain cases of immature or complicated cataract (*vide* p. 316), and in cases where it is necessary to operate in spite of some doubt as to the sterility of the conjunctiva after thorough preliminary treatment. Infection is likely to be more under control after iridectomy than after extraction, and the behaviour of the eye in the first operation is a guide to its probable behaviour after extraction.

In every case of cataract, before operating, the condition of the patient's conjunctiva and lacrymal apparatus is thoroughly investigated as already indicated (p. 470) and full premedication, regional anæsthesia and akinasia carried out.

There are two chief methods of performing cataract extraction: extracapsular and intracapsular. Intracapsular extraction should be employed only by highly skilled operators. Extracapsular extraction with iridectomy ("*Combined Extraction*") is the simplest operation, and "*Simple Extraction*" (with or without a peripheral button-hole iridectomy) should

only be attempted after experience has been acquired by the combined method.

There are many varieties of technique advocated by experienced operators. The simplest technique for combined extraction will be described first, and then a technique for simple extraction. Intracapsular extraction will be only briefly described.

The surgeon stands above the head of the patient, making the section with his right hand for the right eye, and with his

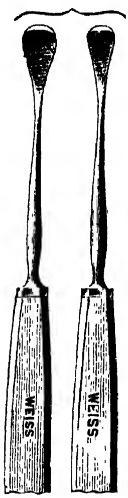


Fig. 260.—Critchett's lens spoon.

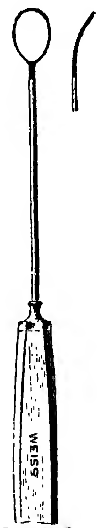


Fig. 261.—Vectis.

Fig. 262.—Arruga's len's expressor.

left hand for the left eye. Some surgeons stand below and at the side for the left eye, and cut away from themselves, using the right hand.

Cataract Extraction with Iridectomy ("*Combined Extraction*"). Instruments required: 5 c.c. syringe, eccentric end with bayonet lock for 1¾ inch needle (for facial nerve block); 1 c.c. syringe with 1¼ inch needle (for Tenon's capsule injection); needle-holder; No. 1 white silk on needle for skin; straight scissors; speculum (Lang's (Fig. 118) or modified Arruga's (Fig. 240)); Graefe cataract knife (Fig. 257); bent

iris forceps; de Wecker's scissors; cystotome (Fig. 258); curette (Fig. 258) or lens expressor (Fig. 262); two iris repositors; [Sinclair's corneal scissors (Fig. 259); lens spoon (Fig. 260) or vectis (Fig. 261); anterior chamber irrigator]. The instruments in brackets are not required for uncomplicated extraction, but must always be ready.

The following description applies to operation upon the right eye.

The speculum having been inserted, the patient is told to look towards his feet and a little outwards. A stitch is passed

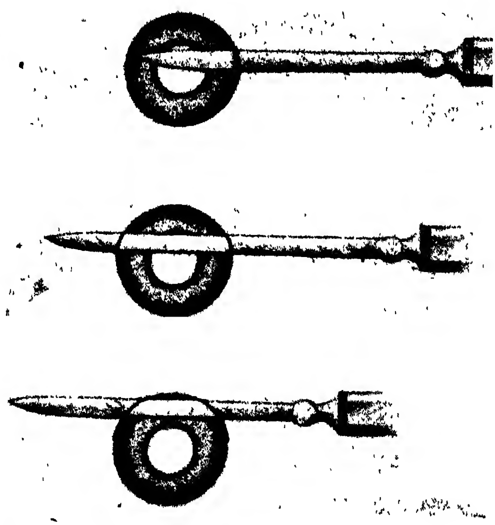


FIG. 263.—*A*. Cataract section, commencing the counter puncture. *B*. Counter-puncture made. *C*. Commencing the section through the limbus.

through the tendon of the superior rectus (*vide* p. 473). The eye is fixed with fixation forceps applied just outside the limbus down and in, below the horizontal meridian of the cornea. The point of the Graefe knife is inserted in the apparent corneo-sclera margin at a point 1 mm. above the horizontal meridian of the cornea, care being taken that the cutting edge is upwards (Figs. 263, 264). It is passed across the anterior chamber to the corresponding spot on the opposite side, the point being aimed to emerge 1 mm. on the corneal side of the limbus. Owing to its apparent displacement forwards by the corneal refraction, it will in this event emerge at the corneo-scleral margin. The

knife is made to cut out in exactly the same manner as in iridectomy for glaucoma (*q.v.*), but following the corneo-scleral margin. A conjunctival flap should be made as in that operation, but it will be narrower at the sides, since the section is farther forwards. It may be made slightly broader above by carrying the conjunctival incision a little upwards after the corneo-sclera has been divided before turning the knife to cut directly forwards. The conjunctival flap is then turned down over the cornea by means of the back of the knife.

The fixation forceps are then removed. If the patient is unsteady the eye is controlled by the superior rectus stitch. The iris forceps are taken in the left hand and the de Wecker's scissors in the right (irrespective of the eye which is being operated upon). The closed points of the forceps are

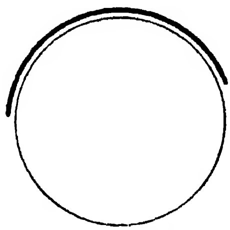


FIG. 264.—Diagram of wound in extraction of senile cataract.

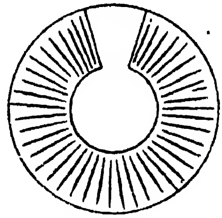


FIG. 265.—Diagram of coloboma in cataract extraction with iridectomy.

inserted at the centre of the wound and passed straight downwards to just above the pupillary margin of the iris. The blades are opened slightly and the iris seized and drawn out of the wound. The piece of iris grasped is cut off with one snip of the de Wecker's scissors, the blades of which are held radially to the iris, *i.e.*, with the points directed upwards (Fig. 243).

The iris repositor is now taken and the iris replaced as in the operation for iridectomy (*q.v.*). Particular care is taken to free it from the angles of the wound, usually an easy task at this stage owing to the support afforded by the lens and the smoothness of the surface of the capsule.

The patient still looking towards his feet, the cystotome is introduced with the cutting edge directed towards the left and slightly upwards. It is passed on until the point is near the lowest part of the pupillary margin. The cutting edge is then directed backwards, the handle is slightly raised, and

the lens capsule is incised vertically as the instrument is slowly withdrawn.

The cystotome is then taken in the left hand, and the curette or the less expressor in the right. The back of the curette or the lens expressor is placed horizontally upon the lower part of the cornea. Gentle but firm pressure is made upon the cornea in a direction backwards and slightly upwards. This causes the nucleus of the lens to be tilted so that the upper edge appears presenting in the wound. The lens nucleus is coaxed out of the wound by repeating the pressure with the curette, but more and more in an upward direction. Meanwhile the lens may be gently helped out by the cystotome in the left hand. As soon as the diameter of the nucleus has passed through the wound, pressure upon the cornea is imme-

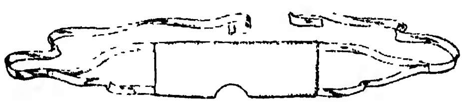


FIG. 266.—Diagram of Moorfields' cataract bandage. The semicircular opening is for the bridge of the nose. The upper and lower tapes are passed above and below the ears. The long single tapes pass behind the head and are brought forward and tied over a pad of wool placed at the upper part of the bandage over the forehead.

diately relaxed. The nucleus is directed down over the cornea into the curette.

If there is much clear soft lens matter, this is coaxed out of the wound by gently repeating the stroking movements on the cornea, or the anterior chamber may be irrigated.

The iris repositor is now again used to free the iris from the angles of the wound. If the iris has been carefully replaced after the iridectomy, as recommended above, little trouble will probably be experienced at this stage. If this step has been omitted it is likely that the nucleus may have jammed the iris into the angles of the wound during its expulsion and there may be great difficulty in freeing it. In addition, the repositor is swept over the whole line of the wound, so as to push back into the anterior chamber any tags of capsule which may be presenting. These are so transparent as to be invisible, and it is extremely important that they should not become incarcerated in the wound.

The conjunctival flap is then turned back to its normal position by the repositor, care being taken that it is not

doubled up. A drop or two of warm atropine solution (1 per cent.) is instilled. The speculum is removed; the lid is raised off the globe and lowered over the eye by the lid stitch, which is then anchored to the cheek by adhesive plaster.

Both eyes are covered by a layer of wide-mesh gauze soaked in refined sterile liquid paraffin, and this is covered by a pad of cotton-wool moistened with normal saline solution. A pad is put over the other eye, and both are bandaged with a many-tailed or a Moorfields' (Fig. 266) bandage. A Cartella shield (Fig. 267) is placed over the operated eye and secured by strapping.

Remarks upon the Operation. The size of the section depends upon the probable size of the nucleus of the lens. It must be remembered that the cortex is soft and broken up; the width of the incision must be slightly larger than the diameter of the nucleus. In black cataract the whole lens is sclerosed, so that a very large section must be made; in these cases it should involve half the circumference of the cornea. More harm is done by bruising the edge of the wound than by having a wound which is unnecessarily large. If the nucleus does not come forward through the wound with moderate pressure of the curette it is probably because the section is too small. The wound should then be enlarged with the probe-pointed bent scissors (Fig. 259). The probe-point of one blade is inserted between the iris and cornea at one angle of the wound, which is then extended by a single snip. The same manœuvre may be repeated at the other angle.

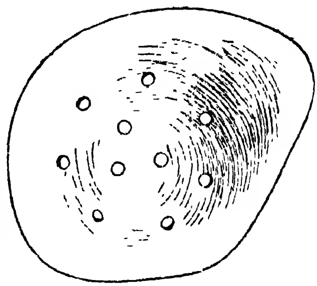


FIG. 267.—"Cartella" shield for right eye.

In making the section the aqueous may escape prematurely, so that the iris floats up in front of the knife. If this happens the knife should be raised as if to lift the eye forward; the aqueous, dammed up in the posterior chamber, can then flow forwards through the pupil and the iris falls back. If this manœuvre fails, the section must be completed in the usual manner, though the iris is wounded, either a hole or a complete coloboma being cut out of it. Cutting the iris causes pain unless Tenon's capsule has been injected and is likely to make the patient flinch, screw up the eye, or completely lose self-

control. Moreover, the coloboma is generally irregular. The accident is usually due to hesitation in pushing the Graefe knife steadily forward or to pressure—often unconscious—exerted on the eye by the fixation forceps. *It is least likely to occur if the passage of the knife through the anterior chamber and the commencement of the section are all part* of a single steady forward sweep of the blade, the handle of the knife being depressed directly the counter-puncture is made. In this manner the broad part of the blade is brought over the iris as quickly as possible. There should, however, be no haste, every movement being made with deliberation but not too slowly.

Old people sometimes have very rigid sclerotics. In these *cases the cornea collapses and becomes saucer-shaped.* This is of no consequence and requires no special treatment.

Sometimes a bubble of air enters the anterior chamber. This also is of no consequence, as it soon becomes absorbed.

The iris is more easily cut off by holding the de Wecker's scissors at right angles to the direction of the forceps, *i.e.*, horizontally (Fig. 244). This makes a larger coloboma, which is unnecessary and has some disadvantages.

Hæmorrhage may occur into the anterior chamber. The blood is derived from the conjunctival flap or from an hyperæmic iris. An attempt may be made to wash it out by irrigation before it clots, or to remove the clot with forceps.

The capsule is divided in all sorts of different directions by different operators. Some surgeons remove part of the anterior capsule by capsule forceps (*vide* p. 488), which has very decided advantages, but has also its own special dangers.

The most serious accident which may occur during extraction is prolapse of vitreous at an early stage. It may be due to inherent weakness of the suspensory ligament, which gives way while the section is being made or the iridectomy done. This cause is most likely to be present in complicated cataracts. In such cases it is a good plan to discard a speculum and rely on the lid stitch to keep the eye open. More commonly loss of vitreous is due to undue pressure on the eye by the fixation forceps. In concentrating all their attention on the section beginners often allow the left hand to dig the fixation forceps into the globe. Special attention must therefore be devoted to the avoidance of this mistake, which has also the lesser disadvantage of forcing out the aqueous and allowing the iris to float up in front of the knife. Escape of vitreous may also be due to pressure with the curette in the

attempt to expel the lens. The necessity of such great pressure is probably owing to the wound being too small or to the capsulotomy having been inefficiently performed. The former contingency has already been dealt with. The latter is overcome by more careful repetition of the capsulotomy. If pressure with the curette causes the vitreous to appear without any sign of the engagement of the edge of the lens in the wound resort must be made at once to scoop extraction. Hence the importance of having the spoon or vectis always in readiness in every case of extraction. The spoon is passed directly backwards into the vitreous so as to make certain that it passes behind the lens. It is then rotated forwards so that the lens is caught between the spoon and the back of the cornea. The lens is kept pressed up against the cornea and is removed by a rapid further rotation of the spoon. Some vitreous is usually lost, but it is imperative that the lens should be delivered, or the eye will almost inevitably be lost. The eye is dressed at once in these cases without any endeavour being made to replace the iris, as any such manœuvre is likely to lead to further loss of vitreous. In spontaneous rupture of the suspensory ligament and in other cases badly managed the lens may sink back into the vitreous. In such cases it is usually futile to attempt to remove it. The eye should be dressed at once, and if the lens floats up into the pupillary area at a later date a further attempt may be made to remove it.

Prolapse of the vitreous after delivery of the lens is less serious, though it increases the tendency to cyclitis, with opacities in the media, and may be followed by detachment of the retina. If much vitreous is lost the iris is always gradually drawn upwards, so that in course of weeks or months the pupil is much displaced and the lower part of the iris stretched (Fig. 245). This condition may also occur from incarceration of the pillars of the coloboma in the wound. It may be necessary to do an iridotomy or some such operation to make an artificial pupil so that vision may be restored.

When the cataract is immature some of the soft lens cortex remains in the eye, so that the pupil is not black but contains greyish masses. Much of the retained lens substance can be removed by stroking the cornea upwards with the curette or lens expressor, repeating the movements used for delivering the lens, but with less pressure. It is usually impossible to remove it entirely in this manner. If it is left in the eye it gradually becomes absorbed, but it has the disadvantages of

tending to irritate the eye and cause slight iridocyclitis, and of leading to the formation of a denser after-cataract. Some surgeons irrigate the anterior chamber with normal saline solution. This method gets rid of the lens substance, but may occasionally set up a mild iridocyclitis, in spite of the strictest antiseptic precautions. Irrigation may be performed with an undine, to the nozzle of which an india-rubber tube is attached, having a flattened canula at the other end. The tip of the canula should be introduced just inside the lips of the wound, and the undine should not be held too high, only a gentle stream of fluid being used.

After-treatment. There is usually some aching and smarting in the eye as soon as the effects of the pantocain wear off. It lasts for four or five hours : hence it is best to operate in the morning so that the patient may have a good night's rest. If the pain interferes with sleep a dose of aspirin will usually relieve it, or a mild bromide draught may be given.

The patient lies quietly upon his back, with the head and shoulders raised. He is directed to avoid all straining. A sneeze may be inhibited by pressure with the finger on the upper lip close to the septum of the nose. *All* patients should have their hands loosely tied to the bed at night, so that they are unable to touch the eyes. Many eyes are lost from neglect of this precaution, for patients often knock or rub their eyes when they are half asleep.

The food must be fluid during the first few days ; no aperient is given for two or three days.

On the day following the operation the bandage is removed, the lids are bathed with warm boric lotion, gently separated, and a drop of sterile 1 per cent. atropine solution instilled. The wound may be inspected, but should not be disturbed more than is absolutely necessary.

On the second day it is examined more thoroughly. The cornea should be bright, and the pupil round and well dilated. Faint greyness in the cornea above (striate opacity, *vide* p. 248) need cause no alarm. Another drop of atropine is instilled. If the pupil is not well dilated on the third day there is probably some trace of iritis, and the atropine should be instilled more frequently, and it may be advisable to resort to hot bathings. In most cases there is no iritis, and after a transient ciliary injection the eye quiets down, so that it is almost free from injection in a week or ten days.

On the second day the unoperated eye may be left unbandaged. If both eyes are kept bandaged too long old people

often become delirious. On the slightest sign of wandering in speech the unoperated eye should be uncovered at once. If this eye is blind or has very defective vision the dressing should be taken off the operated eye, and dark protective goggles worn during the daytime.

It is wise to keep healthy patients in bed for a week. Most cataract patients, however, are old, and extremely liable to hypostatic congestion of the lungs. Such patients should be propped up in bed immediately after operation, and allowed to sit up out of bed soon after. A light dressing is kept on for a week; afterwards smoked glasses are worn until cataract

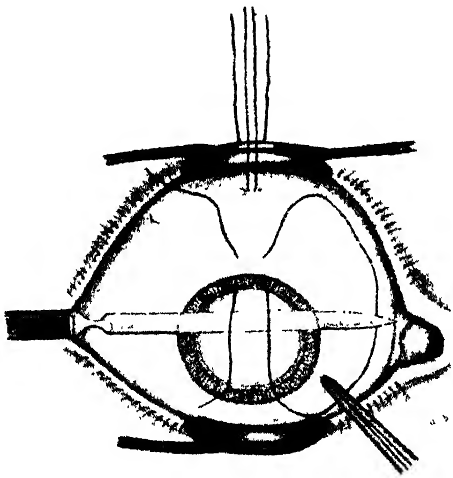


FIG. 268.

glasses can be ordered, *i.e.*, in about six weeks. It is very important that smoked glasses should be worn continually, otherwise the patient is quite likely to develop erythropsia (*q.v.*).

Cataract Extraction without Iridectomy ("*Simple Extraction*"). For this method a corneo-scleral suture may be advisable. Many different types have been advocated but the simplest is that illustrated in Fig. 268. A corneo-scleral eyeless needle armed with 000 black silk is passed through the superficial layers of the cornea transversely for 2·5 mm. at a point about 1·5 mm. on the corneal side of the limbus; it is then passed through conjunctiva and episclera 2·5 mm.

above and exactly opposite and parallel to the direction of the corneal suture (Fig. 268). The corneo-scleral section is made between the corneal and scleral insertions.

The operation is performed in the same manner as that already described except that the iridectomy is omitted.

If the iris impedes the delivery of the nucleus it may be retracted upwards by a blunt iris hook.

When the lens has been extracted an iris repositor is inserted and the iris stroked back into position. The pupil should be quite round: if not, or if the iris tends to prolapse, the periphery is gently drawn out of the wound by iris forceps and

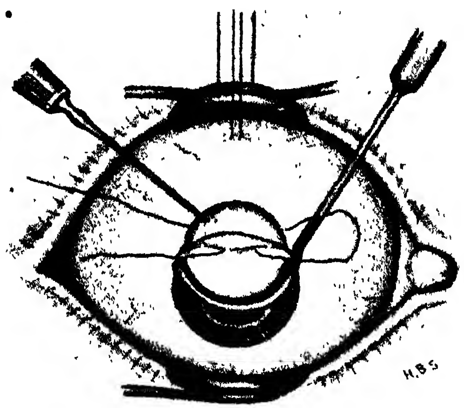


FIG. 269.

the smallest possible fold is snipped off with de Wecker's scissors. The iris is then again replaced with the repositor.

If much soft lens matter remains in the eye, as in immature cataract it may be removed by massage or irrigation (*vide* p. 498), and the iris again replaced.

The corneo-scleral suture is tied firmly enough to coapt the edges of the wound, but not tightly. The conjunctival flap is stroked into position, and the eyes bandaged.

Comparing the two operations, it may be succinctly stated that the chief advantages of *simple extraction* are: Simplicity of the operation, including especially minimum of mutilation, of instruments required, and of instruments introduced into the interior of the eye; optical advantages of a round pupil —minimal dazzling, best visual acuity, and best field; cos-

metic advantage of a round pupil; ease of reposition of the iris; minimal danger of incarceration of capsule in the wound; infrequency of prolapse of vitreous; greater protection of deeper parts of the eye from infection; absence of pain and bleeding from cutting the iris. Of these it may be remarked that the optical advantages are not overwhelmingly manifest. The cosmetic effect is rarely of sufficient importance to outweigh the risks, though occasionally it is a justifiable argument, as in the case of an unsightly unilateral cataract in a young woman, or in a man in whom it forms a bar to obtaining employment. It is doubtful whether prolapse of vitreous is more frequent in combined than in simple extraction. Bleeding from the iris is only very exceptionally of any importance; when there is much bleeding in cataract extraction the blood is derived from the conjunctival flap or from a too peripheral section. The chief disadvantages of simple extraction are: risks of prolapse of the iris; less efficient treatment of the anterior capsule; greater difficulty in removal of soft lens matter; greater danger of ring synechia and secondary glaucoma if iritis should occur.

The chief advantages of *combined extraction* are: greater ease in expression of the nucleus; greater ease in removal of soft lens matter; increased facility in dealing with the anterior capsule, and therefore diminished necessity for subsequent discission; diminished risk of prolapse of the iris; diminished risk of secondary glaucoma. The chief disadvantages are: greater complexity of the operation, including especially need of more instruments and of the introduction of more instruments into the eye, and greater duration of the operation; optical and cosmetic disadvantages; greater danger of incarceration of iris and capsule in the wound; greater danger of post-operative glaucoma; pain and bleeding from the iris.

If there is any difficulty in replacing the iris after a simple extraction, or if the pupil, when the iris is reposed, is not quite circular, an iridectomy should be done at once. Prolapse is liable to occur within the first twenty-four hours after the operation. If it is not treated by an immediate iridectomy a very serious condition will result. The incarcerated iris will fail to withstand the intraocular pressure and a "cystoid cicatrix" will be formed. Visual acuity will be diminished by excessive astigmatism and the eye exposed to grave danger from iridocyclitis, and even panophthalmitis or sympathetic ophthalmia.

Many of the disadvantages of both operations, including the danger of prolapse of iris, are obviated by a modification in which after simple extraction has been performed a small button-hole is made in the periphery of the iris (*peripheral iridectomy*) (*vide supra*). The aperture in the iris is peripheral, so that it is almost entirely hidden by the sclerotic, and in any case is completely covered by the upper lid, while at the same time sufficient drainage from the posterior into the anterior chamber is provided for. Prolapse of iris is less likely to occur than in simple extraction; and it is quite unlikely that the capsule will be left entangled in the wound, a decided drawback to extraction with the ordinary form of iridectomy.

Intracapsular Extraction. In this operation a larger section is necessary than for extracapsular extraction: it should pass across the full horizontal diameter of the cornea. After the section is made a complete or small button-hole peripheral iridectomy is performed—according to the judgment of the surgeon. Capsule forceps (such as Knapp's or Arruga's) are introduced closed to the temporal side of the iridectomy. On reaching the pupil margin the blades of the forceps are moved over the anterior capsule in the sagittal plane and are stopped over the thickest part of the capsule just in front of the equator near the lower edge of the lens. The blades are then opened 3 mm., pressed gently backwards to engage the capsule, and closed. Gentle rotating, zigzag movements are made so as to rupture the suspensory ligament. These movements increase in excursion, and not until the lower edge of the lens is felt to be free and to move forwards is any attempt made to lift the lens and tumble it forwards. The remainder of the manœuvre consists in holding the capsule without pulling on it, and using the lens expressor in the usual way, keeping it just below the lowest part of the lens during delivery. When the lens is in the wound it is important to complete the final stage slowly and deliberately, allowing the vitreous to gravitate and the iris to slide back in place. When the lens has been removed the corneo-scleral suture is tied, sterile drops of eserine (1 per cent.) are instilled, the iris replaced, and the conjunctival flap adjusted.

The chief complications arising after cataract extraction are striate "keratitis" (Fig. 149), incarceration of the iris in the angles of the wound, prolapse of the iris, iritis, iridocyclitis, sympathetic ophthalmia, secondary glaucoma, intraocular hæmorrhage, infection of the wound, panophthalmitis, &c.

Prolapse of Iris is most apt to occur after simple extraction, but may affect either pillar of the coloboma in combined extraction. It usually occurs in the first day or two, but may result later from injury to the eye by rubbing or knocking it, straining, coughing, &c. It must be treated *at once* by excision of the prolapse. As the iris is irritable the operation is painful, and general anæsthesia or retrobulbar local anæsthesia is generally necessary or advisable. The wound is re-opened by insinuating the tip of an iris repositor under the conjunctival flap and gently uncovering the prolapse. The flap is turned down over the cornea, the iris pulled out with iris forceps and snipped off with de Wecker's scissors. The iris is then replaced with a clean repositor and the conjunctival flap brought back into position. A small subconjunctival knuckle of iris can sometimes be replaced by an iris repositor, but it is generally wiser to snip it off.

Delayed Re-formation of the Anterior Chamber may be due to a jagged section, over-riding of the lower lip of the wound, or to no apparent cause. It is much less common in cataract extraction than after glaucoma iridectomy or trephining. In these cases the bandage should be very lightly applied or discarded, dark protective goggles being worn in the daytime, and a light bandage with wire or cartella shield at night.

Delayed Healing of the Wound is more likely to occur with a purely corneal section, such as some surgeons prefer. The patient should be kept in bed until it is firmly healed, unless this is specifically contraindicated (*vide* p. 499). Delay in healing is, however, generally due to incarceration of iris or capsule in the wound. If this amounts to an actual prolapse it must be operated upon (*vide supra*), but it may be very insidious. In either case the result may be the formation of a *cystoid cicatrix*, part or the whole of the scar slowly and gradually becoming more and more ectatic. The eye should be carefully examined to see if the pupil or either pillar of the coloboma is drawn up, or if capsule can be seen in the wound. A fully developed cystoid scar should be left alone, though it causes much astigmatism and is liable to give rise to secondary infection, or iridocyclitis and even sympathetic ophthalmia.

Expulsive Hæmorrhage is fortunately rare. It occurs during or soon after operation in old people with arteriosclerosis or some diathesis, such as diabetes. There is sudden severe pain, and on removal of the dressings the wound is found to be gaping and filled with blood clot, vitreous, &c. The eye is always lost and should be excised. This may be necessary in order to stop the bleeding, the socket being then packed and firmly bandaged.

Septic Infection may occur in spite of all precautions, especially in diabetic patients. It is most commonly due to the pneumococcus, but may be caused by the streptococcus, staphylococcus aureus or even albus, and many other organisms. It usually occurs from the twelfth to thirty-sixth hour after operation. There is severe aching pain, due to the accompanying acute iritis. On removing the dressings the upper lid is œdematous. When the lids are separated tears gush out and there is muco-pus in the conjunctival sac. The lids should be separated gently, if necessary with retractors. The cornea is then seen to be dull and hazy, especially in the upper part, the lip of the wound being yellow. Almost invariably the infection spreads rapidly. Intense iritis is set up, the pupil and coloboma become filled with exudate and an hypopyon appears. Finally, the vitreous becomes infected and panophthalmitis leads to the destruction of the eye.

Treatment is seldom of any avail, but must be applied quickly and energetically. The anterior chamber should be irrigated with a solution of pure penicillin (1,000 units per c.c.) which may be repeated on subsequent days; subconjunctival injections may also be given (*vide* p. 696). In addition sulphonamide treatment (*vide* p. 695) may be tried. A staphylococcic or polyvalent vaccine should be given and an autogenous vaccine prepared and administered as soon as possible.

Iritis in mild degree probably occurs in all cases of cataract extraction. In more pronounced form it is specially associated with retained lens matter (*vide* p. 498) and diathetic states, such as diabetes, rheumatism, gout, &c. The worst cases occur with acute septic infection. Intermediate in severity are cases of plastic iritis due to infection by less virulent organisms or in patients with greater resistance to bacterial invasion. More insidious than any are cases of continued irritability of the eye with mild iritis. In these, spots of " k.p." are found upon the back of the cornea, so that there is also cyclitis. Both in these cases and in the cases of plastic iritis there is grave danger that the condition is really sympathetic ophthalmia. Hence it is very essential in all cases of cataract extraction to inspect the cornea most carefully with oblique illumination and the loupe and to do so frequently, especially if there is an unusual degree of flushing and lacrymation on exposure to light. The other eye must also be carefully watched. If there is " k.p." no needling operation must be undertaken until the eye has quieted down and remained quiet for many weeks. It is sometimes difficult to distinguish minute spots of lens substance on the back of the cornea from true " k.p."; they soon become absorbed.

Detachment of the Choroid. *Vide* p. 348.

Secondary Glaucoma may set in after cataract extraction. It is probably usually due to peripheral anterior synechia and incarceration of capsule in the wound (*vide* p. 282). Sometimes it is due to the anterior chamber being lined with epithelium. In these cases there has been delay in healing and the conjunctival epithelium has grown down into the anterior chamber and spread over the surface of the iris, lens capsule and cornea. These cases are practically hopeless and cannot be diagnosed clinically with certainty. Sometimes secondary glaucoma follows needling of the after-cataract. It is usually then attributed to vitreous extending into the anterior chamber and interfering with filtration. It is doubtful if this is the true explanation. These cases usually do badly. As regards treatment, where there are definite adhesions of capsule an attempt may be made to divide them. These cases afford the best prognosis. In the more obscure cases a cyclodialysis is usually the operation of choice, but if it fails the eye should be trephined.

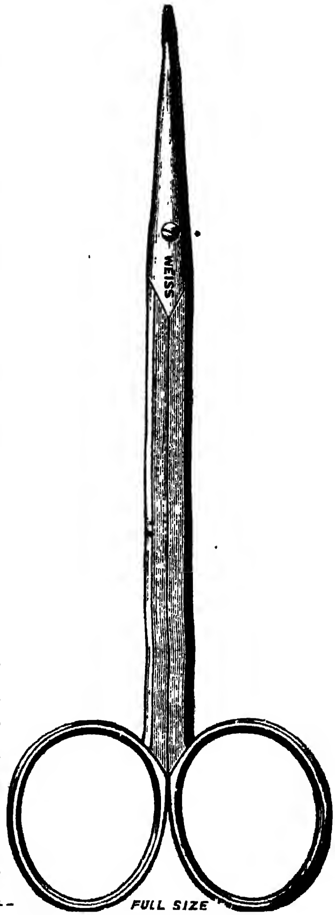


FIG. 270.—Tenotomy scissors.

EXCISION OF THE EYEBALL

The appalling tragedy of removing the wrong eye is known to have occurred. The accident is most likely to happen when there are no superficial evidences of disease, as for example in excision for sarcoma of the choroid. It is well to mark the forehead above the eye to be removed before the anæsthetic is administered.

A general anæsthetic or deep local anæsthesia is required. The operation can be performed almost painlessly under novocain (*vide* p. 471), and this method is sometimes advisable in old people with diseased arteries, or in patients with heart disease.

Instruments required: speculum (preferably Lang's), two pairs of fixation forceps, tenotomy scissors (Fig. 270), strabismus hook (Fig. 271), blunt-pointed scissors—straight or curved on the flat (Fig. 275).

The surgeon stands above the patient.

The conjunctival sac having been douched and the speculum inserted, the surgeon seizes the conjunctiva just outside the limbus at the highest part of the cornea. The conjunctiva is incised here with the tenotomy scissors. The point of one blade of the scissors is passed under the conjunctiva and pushed on as far as possible round the cornea. By carrying the point out under the looser bulbar conjunctiva it may be taken a third of the distance round the circumference; the edge is then brought close up to the limbus before the conjunctiva is divided. Still fixing the eye in the same position the manœuvre is repeated on the other side of the cornea. Finally, the portion below the cornea is divided. The conjunctiva should be divided completely round the cornea, and close to it, in three or four cuts.

Fig. 271.—Strabismus hook, flat Moorfields' pattern.

The peripheral edge of the cut conjunctiva is then taken up by the forceps, and the bulbar conjunctiva is separated from the globe as far back as the equator in all directions by a series of small snips, the blades of the scissors being kept flat in close contact with the eyeball. In this manner the capsule of Tenon is simultaneously opened.

The tenotomy hook is then taken in the left hand, the scissors being retained in the right. The recti muscles are taken up one by one and divided close to the globe. It is well to begin with the superior rectus, since it is the most difficult to get at, especially if the other recti have been previously divided. The tendon of the external rectus of the right eye or the internal rectus of the left eye should be left long. The obliques are found by passing the hook farther back and carrying it round close to the globe.

The speculum is then taken and held widely open and pressed back into the orbit. If the muscles have been

properly divided the globe springs forwards between the blades of the speculum. The other pair of scissors is now taken in the right hand. The appropriate long tendon stump is then seized firmly with toothed forceps and the globe pulled forward and rotated towards the left side of the patient so that the optic nerve is easily reached. The points of the closed scissors are passed into the orbit—to the outer side of the eye on the right side, to the inner on the left. The optic nerve is felt for with the closed scissors: it is easily recognised. The scissors are withdrawn a short distance, opened, and the blades pushed down, one on each side of the nerve, which is then divided. The sensation of dividing the nerve is unmistakable. The eyeball can then be freely drawn forwards. There are probably some remnants of the obliques still attached to the globe. These are divided close to the eye. If the bleeding is profuse the inside of the muscle cone is packed with ribbon-gauze wrung out in hot saline: pressure is kept up for two or three minutes. The edges of the conjunctiva are then pulled together with the fixation forceps or better drawn together by a continuous silk suture, the lids closed, and the dressing applied. The latter should consist of a small spherical pad of gauze, then a round flat pad of sterilised or cyanide gauze, then a thick round pad of sterilised wool. The bandage is applied with a firm pressure. The patient is kept in bed for one or two days. The suture is removed after forty-eight hours.

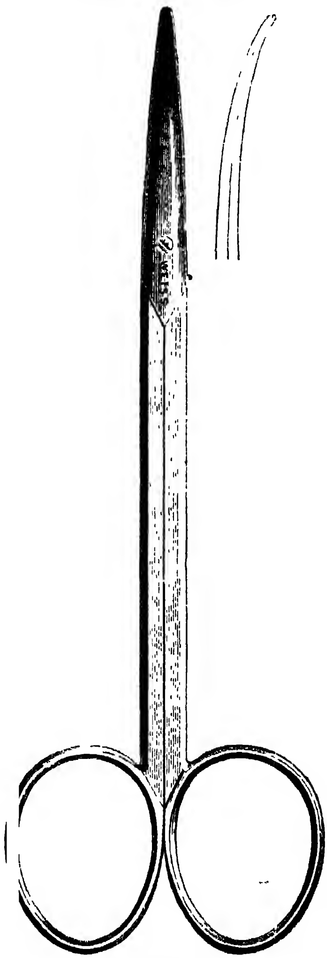


FIG. 272.—Excision scissors.

If the globe is perforated and collapsed excision is more difficult. The rupture should be closed by sutures before proceeding to excise the eye. In children also excision is difficult. The orbit is small in proportion to the size of the globe. Here the manœuvre with the speculum to dislocate the ball forwards is often unsuccessful, and strong pressure may burst the eye. It is best levered out with the points of the excision scissors.

It is very easy to cut the sclera instead of the nerve, especially with curved excision scissors. I always prefer straight scissors; indeed, straight instruments should always be used in preference to curved whenever it is possible, because it is much easier to judge the position of the point. Straight scissors are particularly indicated when the nerve has to be cut long, as in excision for retinoblastoma and sarcoma of the choroid. Special precautions must be taken in excision for panophthalmitis (*vide* p. 463).

Evisceration of the eyeball is recommended only in some cases of panophthalmitis (*q.v.*). Some surgeons use it in anterior staphyloma and other conditions. Although the stump affords a good foundation for an artificial eye, the operation is not to be recommended in these cases; it has been followed by sympathetic ophthalmia.

Excision, with the introduction of a glass globe in Tenon's capsule is a good method in carefully selected cases and affords admirable support to an artificial eye. The eye is much more movable than after simple excision, so that the simulation of a real eye is more exact. Care must be taken during the excision to keep close to the eyeball and to injure Tenon's capsule as little as possible. The recti muscles are drawn together over the small glass globe by a buried purse-string suture of catgut (000,000, ten day). The conjunctiva is sutured by a superficial purse-string suture. There is some inflammatory reaction, with chemosis, but little or no pain. If the operation has not been well performed, the globe may slip into the orbit, becoming loose, usually beneath the lower lid. If this occurs it must be removed and the case treated as an ordinary excision. More recently globes of plastic or spheres of tantallum mesh have been used into which an artificial eye may be inset: such a procedure is technically difficult but if successful the cosmetic results are excellent.

An *artificial eye* should not be worn less than six weeks after excision. A small eye is first worn for an hour or two a day until the conjunctiva becomes used to the foreign body.

Eight or nine weeks after the operation a full-sized eye may be worn. It is taken out at night, carefully washed, and kept in water. Various types of artificial eye are made, either of glass or plastic. Of the former, Snellen's "reform" eye gives a better cosmetic effect after the ordinary excision than the old shell, which may be used when there is good support, as after insertion of a globe in Tenon's capsule. Glass eyes tend eventually to become rough, in which event they must be discarded at once. This usually occurs from chemical action of the secretion upon the enamel in about a year, so that in any case a new glass eye should be obtained every year. Plastic eyes are more comfortable to wear than glass eyes and have the great advantages of being unbreakable and of adapting themselves readily to the temperature of the socket —a great advantage in intense cold. The methods of insertion and removal of the artificial eye should be learnt by every surgeon by seeing it done.

Contracted Socket is the result of injury, faulty excision cellulitis in the orbital tissues, or the continued wearing of a rough artificial eye. The first three causes lead to the formation of dense cicatricial bands across the socket, rendering the wearing of a prothesis impossible. The last cause usually results in obliteration of the lower fornix, so that the eye cannot be kept in place.

Contracted sockets are very difficult to remedy. It is easy to divide the bands and make a new groove to hold the eye in position, but unless the wounds become covered with epithelium the edges heal together and no improvement is produced. A thorough dissection of all fibrous bands should be made, and the raw surface covered by a Stent mould. The Stent is cooled *in situ* by drops of cold saline and removed. A clamp is applied to the lower lip, which is then everted to expose its inner aspect. The Stent mould is placed over this, and a graft of mucous tissue nearly twice the size is cut. The submucous tissue is dissected off and the graft sutured in position. The Stent is secured in place over this by mattress sutures; the eyelids are closed and covered with a dressing of tulle gras gauze wrung out in saline, and a pad and bandage.

The greatest difficulty is to restore the lower fornix. Maxwell's operation is the best for this purpose if the lower lid is uninjured. It is simple, but difficult to describe. An elliptical area of skin is marked out on the lower lid. The upper incision is carried through into the socket, in the position of the new fornix. The flap is about 8 mm. broad

in the centre. It is dissected up at the edges all round, but a central elliptical pedicle is carefully retained. The flap is tucked through into the orbit; the upper edge is sutured to the posterior lip of the conjunctival wound, and the lower edge to the anterior lip. The gap in the skin is then closed with sutures, and a glass shell is inserted in the socket. The pedicle ensures the vitality of the flap and also keeps the new fornix depressed to the level of the orbital margin. If much ectropion results the scar can be re-opened at a later date and a Wolfe graft of sufficient size inserted and sutured in position with eyeless needles and fine silk or gossamer horsehair. A moulded Stent is placed over the graft and retained by mattress sutures and a strip of gauze secured to the adjacent skin by mastisol.

When there is a good upper fornix a simpler procedure may be tried. The conjunctiva is extensively undermined through a temporal vertical incision sufficient to admit a pair of blunt-ended scissors. When the conjunctiva covering the floor of the socket is free two or three mattress sutures are passed through it in the part designed to form the lower fornix (Fig. 342), and carried down through the periosteum of the intraorbital margin, the orbicularis and skin. They are then tied over pieces of rubber tubing.

In bad cases of contracted socket it is necessary to dissect away all the remaining conjunctiva and fibrous tissue. As large a Stent mould as the socket will retain is then fashioned and its centre is perforated by a hole 2 mm. in diameter for the escape of discharge. An ample Thiersch graft is cut by Humby's knife and wrapped round the Stent with its raw surface outwards. This is inserted into the socket and the lids are united by a central tarsorrhaphy.

SECTION IV

ERRORS OF REFRACTION AND ANOMALIES OF ACCOMMODATION

CHAPTER XXIII

Retinoscopy

(Before reading this section the student should revise his knowledge of the optical conditions of the eye and the methods of testing visual acuity by again reading Chapters III., IV., VII., and IX.)

It has been already pointed out that the condition of the refraction of an eye can be estimated in various ways. The systematic examination of the visual acuity will in most cases *indicate* the absence or the nature of any error of refraction. The examination with the mirror at a distance of 1 metre also indicates the refractive condition by the visibility of retinal vessels and the direction of parallactic displacement; as will be shown below, this method may be made to give very accurate estimation of the exact refraction. The indirect method also indicates the refractive condition by the apparent change in size of the disc when the large lens is moved away from the eye. By the direct method the condition of the refraction can be accurately measured if the surgeon has acquired the ability completely to relax his accommodation.

Retinoscopy, or, more correctly, *skiascopy* or *the shadow test*, is the most accurate means at our disposal of estimating the condition of the refraction objectively. It depends upon the fact, first pointed out by Bowman, that when light is reflected from a mirror into the eye the direction in which the light travels across the pupil varies with the condition of refraction of the eye. If the light is thrown into a myopic eye from a concave mirror at a distance of 1 metre the light, or, what is easier to observe, the shadow, moves across the pupil when the mirror is slowly tilted in the same direction

as that in which the mirror is moved (Fig. 273). If a plane mirror is used, the other conditions remaining the same, the shadow will be seen to move in the opposite direction to the movement of the mirror. If the eye is hypermetropic the direction in which the shadow moves is the opposite of that with the myopic eye. If the eye is emmetropic or has only a very low degree of myopia no shadow will be visible; the pupil will be either completely illuminated or completely dark.

The light seen in the pupil is the blurred image of the illuminated area of the fundus as seen by the observer when

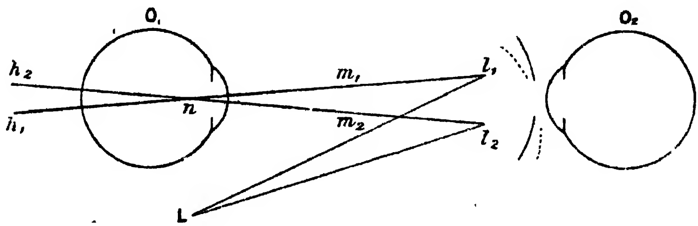


FIG. 273.—Diagram of retinoscopy with a concave mirror. O_1, the observed eye; O_2, the observer's eye. The image of the source of light is formed at l_1 (the immediate source of light) by the mirror. If O_1 is hypermetropic a virtual image of l_1 is formed on the line l_1 n, passing through the nodal point n, as at h_1. If O_1 is myopic a real inverted image is formed as at m_1. If the mirror is tilted downwards, as shown by the dotted line, l_1 moves to l_2, h_1 to h_2, and m_1 to m_2. This shows that the shadow moves in opposite directions in hypermetropia and myopia.

he accommodates for the observed pupil. The shadow is merely the image of the edge of the illuminated area.

Imagine a point of light in front of an eye, the pupil being dilated and the accommodation paralysed by atropine (Fig. 274). The divergent rays which enter the eye are made convergent by the refractive media, so that a circular area of the fundus, varying in size according to the refraction of the eye, is illuminated. If the point of light moves upwards, the light on the retina will move downwards.

Now consider the rays of light which are reflected from the illuminated area. In the hypermetropic eye they will be divergent, as if they came from a point behind the eye. This far point, corresponding with the illuminated area, will move in the same direction, *i.e.*, downwards. Now imagine an observer, placed in front of the eye, to look towards a point of

light situated at the position of the far point, but to accommodate for the position of the observed pupil. He will see a circle of light with a blurred margin, not a point, because he is not accommodating accurately for the far point. When the illumination on the retina moves down, the circle of light which the observer sees will appear to move down also (Fig. 274).

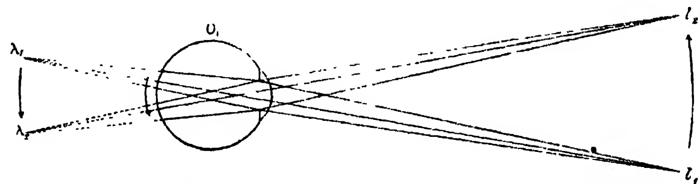


Fig. 274.—Showing the course of incident rays and field of illumination of the fundus in hypermetropia. l_1 forms a virtual image at λ_1, l_2 at λ_2. The field of illumination is determined by the pupil of O_1.

Again, consider the rays of light reflected from the illuminated area on the fundus of a highly myopic eye. They will be convergent and will cross at a real point in front of the eye. This far point, corresponding with the illuminated area, will move upwards when the illuminated area moves downwards. An observer placed in front of the eye and farther from it than the far point, if he looks towards the far point but accommodates for the observed pupil, will see a circle of light with a blurred margin. When the illumination on the retina moves

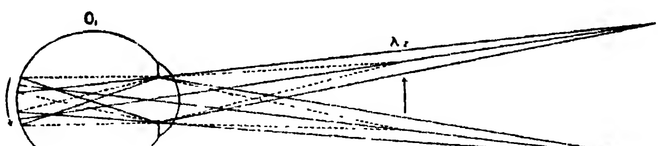


Fig. 275.—Showing the course of incident rays in myopia.

down, the circle of light which the observer sees will move up, *i.e.*, in the opposite direction to the movement in the case of the hypermetropic eye (Fig. 275).

Now suppose that the observer's eye is one metre in front of the observed eye, and that the latter has 1 D of myopia. In this case the far point of the observed eye will be at the situation of the observer's eye, say at the level of his pupil (Fig. 276). In this case a very slight movement of the light

on the observed fundus will throw the image at the far point off the observer's eye altogether. In other words, the observed pupil will appear to be completely bright or completely dark.

If, again, the observed eye is emmetropic, its far point will be at infinity. We may regard it as being infinitely far behind the observed eye. Here, again, there will be scarcely any shadow, though in reality there is a very faint shadow moving in the same direction as for the hypermetropic eye.

The above is a simple explanation of the theory of retinoscopy. The question of the type of mirror is an entirely subsidiary one. It merely has to do with the direction of movement of the immediate source of light, *i.e.*, the point of light in front of the eye which has been considered above. The image of a real light behind the patient's head, formed by a concave mirror, is situated in front of the mirror. If the

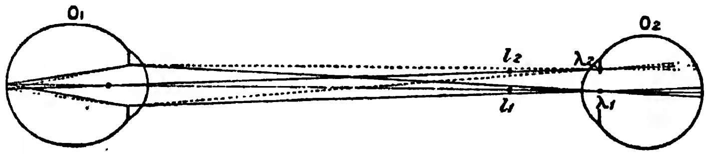


FIG. 276.—Showing the course of the emergent rays at the point of reversal. So long as λ_1 is in the pupillary area of O_2, the pupil of O_1 appears uniformly illuminated, and there is no shadow. Directly λ_1 passes to λ_2 the whole of the light is cut off, so that the pupil of O_1 becomes completely dark.

mirror is tilted up, the image moves up. The image of a real light behind the patient's head, formed by a plane mirror, is situated as far behind the mirror as the light is in front of it. When the mirror is tilted up, the image moves down.

Hence under the actual conditions of retinoscopy with a plane mirror, when the mirror is tilted to the right the immediate source of light moves to the left, and—

(*a*) In the hypermetropic eye, the circle of light on the fundus moves to the right, and the shadow seen in the pupil moves to the right;

(*b*) In the myopic eye (above - 1 D) the circle of light on the fundus moves to the right, and the shadow seen in the pupil moves to the left;

(*c*) In the myopic eye of — 1 D there is no shadow;

(*d*) In emmetropia and myopia of less than — 1 D there is a very faint shadow moving to the right.

Stated as a mere guide to practice, with the plane mirror the shadow moves in the same direction as the mirror in

hypermetropia and in the opposite direction in myopia above one dioptre; in myopia of one dioptre there is no shadow and in emmetropia and myopia of less than one dioptre there is a very faint shadow moving in the same direction as the mirror.

In actual retinoscopy the whole of the image of the illuminated area of fundus cannot be seen at once; the shadow is part of the circumference. In high degrees of ametropia the shadow has a distinctly curved border, it is very dark, and it moves very slowly (Fig. 277). In low degrees of ametropia the border of the shadow looks straight; it is faint, and it moves very rapidly.

The movement of the shadow, being a purely optical phenomenon, is, of course, independent of the cause of the ametropia. Consequently, in astigmatism, if one axis is hypermetropic and the other myopic (mixed astigmatism) the shadow moves in opposite directions in the two meridians. Often the periphery of the cornea is flatter than the centre; correction of the refraction of the central part, which is the more important, will then differ from that of the peripheral part. These variations produce very puzzling shadows in many cases.

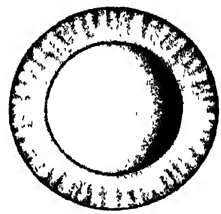


FIG. 277.

Retinoscopy is applied to the estimation of refraction by placing correcting lenses in front of the eye and noticing the effect upon the shadow. When the shadow has completely disappeared we know that the eye has been made myopic D 1 if the surgeon is at one metre from the patient.

Retinoscopy is conducted in a dark room. The light is placed behind and above the patient's head. The surgeon sits at one metre from the patient. The patient wears a trial frame; the eye not under observation is covered by a screen. A mydriatic should be used by all but skilled observers, and is necessary to them in many cases. A plane mirror should be used. The patient looks at the observer's forehead.

The light is reflected into the eye, and the mirror is slowly tilted from one side to the other. The direction in which the shadow moves is noted. The horizontal meridian should be observed first, then the vertical. If the shadow appears to swirl round, not moving in the same meridian as the mirror, the eye is astigmatic, and the mirror is not moving in a direction which corresponds with either axis. A direction of move-

ment can then be found in which the shadow will move either directly with or against the mirror ; this is one of the principal axes of the astigmatism. The other axis is at right angles (regular astigmatism).

If the shadow moves with the mirror a low convex glass is put in the frame in front of the eye. If the shadow still moves in the same direction a stronger convex glass is used, and so on until no shadow can be seen. A still stronger convex glass is placed in the frame. The shadow now probably moves against the mirror. We now know that the refraction has been over-corrected. The point at which there is absolutely no shadow—the point of reversal—is somewhere between the last two lenses, and we know that at that point the refraction of the eye *plus* the lens is equivalent to one dioptre of myopia. If, for example, the shadow can still be seen to move with the mirror with + 4 D lens in the frame, and moves against it with + 5 D, we shall not be far wrong in considering that the point of reversal is + 4·5 D. A + 4·5 D lens would therefore make the eye one dioptre myopic. The actual refraction is therefore + 3·5 D. If there is no astigmatism the patient ought to be able to read 6/6 with this lens. If he is under atropine a further correction must be made before glasses are ordered. Atropine not only paralyses the ciliary muscle, but also inhibits the physiological tone of the muscle. This is found by experience to be equivalent to about 1 D of accommodation. Hence the glass which should be ordered to correct the distant vision after the mydriatic has passed off is + 2·5 D.

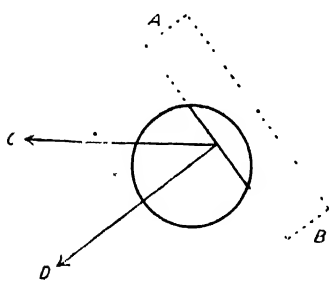


Fig. 278.

Similarly for spherical myopia. Supposing — 4 D eliminates the shadow against the mirror and — 4·5 D gives a distinct shadow with the mirror, we know that — 4·25 D will leave the eye with still — 1 D. Hence the refraction under atropine is — 5·25 D. The correction for atropine gives — 6·25 D as the lens which corrects distant vision without a mydriatic. The tone of the ciliary muscle is often less in myopia than in hypermetropia : since myopia should be under- rather than over-corrected, it is wiser to order very little more than the atropine correction, *e.g.*, 5·5 D in the above example.

In astigmatism each principal meridian is corrected separately. When one meridian is approximately corrected the shadow assumes the shape of a band. The edge of the band is parallel to the axis of the corrected meridian. Even if the light is not moved in a direction accurately at right angles to this meridian the shadow still seems to move in the same direction. This is due to an optical illusion. If a straight edge, A B, is placed obliquely behind a circular hole in a card and is then moved horizontally in the direction of the arrow C, it will appear to be moving in the direction of the arrow D at right angles to its own edge (Fig. 278). The shadow is most sharply defined if the mirror is moved at right angles to its edge, *i.e.*, at right angles to the corrected meridian.

The results are usually recorded thus (Fig. 279), the directions of the lines indicating the directions of the axes :—

The numbers should represent what the surgeon believes to be the refraction of the eye under the mydriatic, not numbers

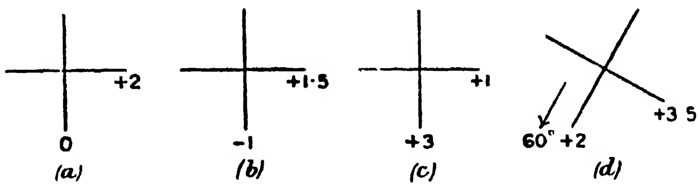


Fig. 279.

to which further modifications have to be made, *e.g.*, the actual lenses used. In the examples given (*a*) is a case of regular simple hypermetropic astigmatism according to the rule; (*b*) regular mixed astigmatism according to the rule; (*c*) regular compound hypermetropic astigmatism against the rule; (*d*) regular compound hypermetropic astigmatism with oblique axes. The exact direction of the axis in astigmatism is usually determined by subjective trial. In children it may be found by placing the requisite cylinder in the trial frame and rotating it until no shadow can be observed in any direction. In the examples given the correcting lenses required would be (*a*) + 2 D cylinder, axis vertical; (*b*) — 1 D sphere combined with + 2·5 D cylinder, axis vertical, or + 1·5 D sph. ⊂ — 2·5 D cyl., axis horizontal; (*c*) + 1 D sph. ⊂ + 2 D cyl., axis horizontal, or + 3 D sph. ⊂ — 2 D cyl., axis vertical; (*d*) + 2 D sph. ⊂ + 1·5 D cyl., axis 60° down and in, or down and out, according as it represents the left or the right eye.

To avoid ambiguity in ordering glasses the axes of cylinders should be uniformly numbered according to the method commonly used by British opticians (Fig. 280).

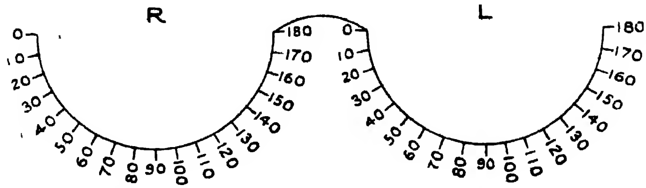


FIG. 280.—Standard numeration of axes of cylinders.

The shadows in regular astigmatism are not always easy to correct, owing chiefly to differences in curvature of different parts of the cornea. Usually the periphery of the cornea is flatter than the centre. The centre of the pupillary area will then be corrected by a different lens from the periphery, especially with the dilated pupil. From this cause various conflicting shadows may be seen, the commonest being the so-called "scissors" shadows, where two shadows appear to meet each other and cross as the light is moved in a given direction (Fig. 281). These difficulties are diminished with the undilated pupil, and an experienced retinoscopist can obtain reliable results under these conditions with an intelligent patient. Relaxation of the patient's accommodation without a mydriatic is best obtained if the retinoscopy is done in a large dark room and the patient is told to look "right across the room." In conical cornea a triangular shadow with its apex at the apex of the cone, *i.e.*, usually slightly below the centre of the cornea, appears to swirl round its apex as the mirror is moved.

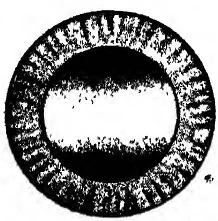


FIG. 281.

Retinoscopy is most valuable in determining accurately the amount of astigmatism, *i.e.*, the difference between the two meridians.

In irregular astigmatism the shadows move in various directions in different parts of the pupillary area; they cannot be accurately corrected by spherical or cylindrical lenses, but some improvement of vision may be obtained.

In conclusion, a word of warning must be given. The correc-

tion of a given refraction by retinoscopy may be very easy or very difficult. A vast number of refractions should have been carefully corrected and confirmed by subjective tests before a surgeon should consider himself justified in ordering glasses without supervision from an expert.

CHAPTER XXIV

Errors of Refraction

Myopia, or "short sight," is that dioptric condition of the eye in which, with the accommodation at rest, incident parallel rays come to a focus anterior to the light-sensitive layer of the retina. Myopia may be due theoretically to any of the following conditions:—*A*. Abnormal length of the eye—*axial myopia*. *B*. Abnormal curvature of the refracting surfaces—*curvature myopia*: (*a*) too strong curvature of the cornea; (*b*) too strong curvature of one or both surfaces of the lens. *C*. Abnormal refractive indices of the media—*index myopia*: (*a*) too high index of the cornea or aqueous; (*b*) too high total index of the lens, due to (*a*) too high index of the nucleus; (β) too low index of the cortex; (γ) both these causes; (*c*) too low index of the vitreous. *D*. Abnormal position of the lens, *i.e.*, displacement forwards. *E*. A combination of the above abnormalities.

It has been proved that emmetropic eyes may differ in length by as much as 1—2 mm., and that the radius of curvature of the cornea may vary from 7—8 mm. Emmetropia therefore results from the integration of all the variables mentioned in the previous paragraph. Statistically one might expect its incidence to resemble the Gaussian frequency curve, but since the full development of emmetropia is never present normally at birth the curve will have a certain "skew deviation." Almost inevitably some cases will fail to reach emmetropia and remain hypermetropic, while others will proceed too far and become myopic. Of these the former are by far the more numerous. I am of opinion that many cases of low myopia come into this category. They cannot in the true sense be regarded as pathological, and they may be expected to remain permanently unprogressive (*developmental myopia*). I have observed many cases of low myopia, with normal fundi, which have remained stationary for many years.

There is no question that increased length of the eye is the most important factor in the high degrees. It is not improbable that the other factors are of more importance than is commonly thought in the lower degrees. Curvature myopia

occurs commonly as a factor in astigmatism, but is rare as a cause of spherical myopia, and is then associated with disease of the cornea—conical cornea. Index myopia is seldom seen clinically, but it accounts for myopia as a premonitory symptom of senile cataract, when it is due to increased refractive index of the nucleus of the lens; it also accounts for myopia in some cases of diabetes, with or without cataractous changes in the lens.

The increase in length of the eye affects the posterior pole and the surrounding area; the part of the eye anterior to the

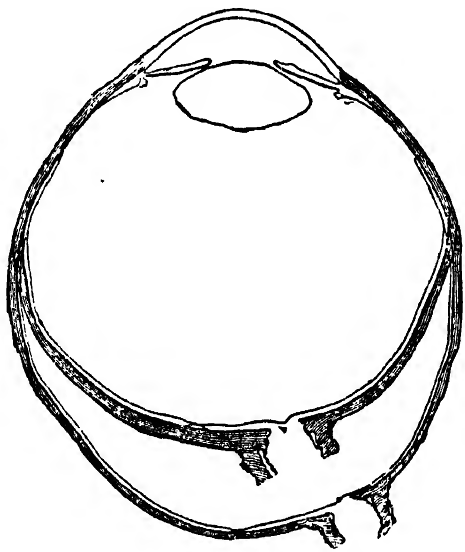


FIG. 282.—Horizontal sections of emmetropic and myopic eyes from the same patient superposed, showing the identity of the pre-equatorial regions. (Heine.)

equator may be absolutely normal (Fig. 282). In most cases the myopia is of low degree, *i.e.*, up to 5 or 6 D (simple myopia). Less commonly the error reaches a considerable degree in childhood or early youth and increases steadily up to twenty-five or more, finally amounting to 15 to 25 D or more (*progressive myopia*). It is impossible, clinically or pathologically, to draw a distinct line of demarcation between the two forms.

In low myopia the only symptom may be indistinct distant vision. In other cases and in high myopia there is often, in addition, discomfort after near work, due largely to dispro-

portion between the efforts of accommodation and convergence (*vide* p. 549). The eyes are unduly sensitive to light. Black spots are seen floating before them, and sometimes flashes of light are noticed; the latter may occur irrespective of any tendency to detachment of the retina (*vide* p. 378). In very high myopia the eyes are prominent, the pupils are large, and the anterior chamber appears deeper than normal, probably only owing to the dilatation of the pupil. There may be an apparent convergent squint due to a large negative angle γ (*vide* p. 571). A true divergent strabismus may be found, either concomitant or affecting only one eye. Vision may be very poor, even with correction; scotomata may be present, both central and peripheral.

Ophthalmoscopically, in low myopia there may be a quite normal fundus; the optical defect will of course be noticed, especially on examination by the direct method. In the majority of cases of moderate myopia there is a "myopic crescent" (Plate XVIII., Fig. 2). This is a white crescent at the temporal border of the disc; very rarely it is nasal. In higher degrees of myopia it may extend to the upper and lower borders, or a complete ring may be formed round the disc. The crescent is occasionally absent even in cases of high myopia.

The bulging at the posterior pole in high myopia is called a posterior staphyloma. It is distinguishable clinically only by its optical and pathological effects. The term should not be used as a synonym for myopic crescent, as is often done. Optically posterior staphyloma causes the high error of refraction, and the edges may be actually visible by the indirect method owing to the presence of a crescentic shadow two or three disc diameters to the temporal side of the disc and concentric with it and to the change in course of the retinal vessels (staphyloma posticum verum). Pathologically, posterior staphyloma causes degenerative changes in the choroid and overlying retina: these are commonly described as "myopic choroiditis," but this term should be abandoned since the condition is non-inflammatory and should be called myopic choroido-retinal atrophy. The changes are generally limited to the posterior pole and the surrounding area (Plate XVIII., Fig. 2). Small yellowish, white, or pigmented spots, and not infrequently white branched lines, usually horizontal, are found at and around the macula. The spots coalesce, forming irregular areas which may extend to the disc. Patches of choroidal atrophy are common near the

disc; they may fuse with each other and with the myopic crescent so as to form a ring round the disc. Small foci occasionally occur at the periphery. Hæmorrhages in the macular region are generally described in high myopia. I believe them to be rare; the appearance of small hæmorrhages is very nearly simulated, but the spots undergo no change for an indefinite time; they are probably due to bunches of dilated capillaries, usually choroidal, rendered visible by rarefaction of the retinal pigment layer. The retinal pigment epithelium often loses much of its pigment in high myopia, so that the fundus is tigroid, and the choroidal vessels are well seen; this condition is not inconsistent with good vision.

Black specks in front of the eyes are often complained of in myopia. Dusty vitreous opacities may be visible with the

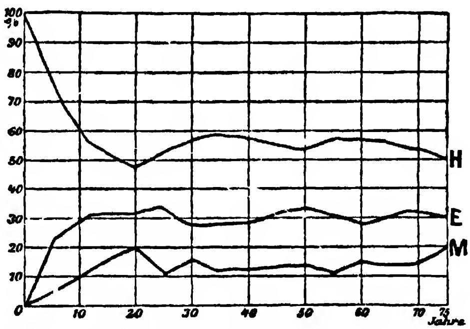


FIG. 283.—Age incidence of ametropia. (Herrnheiser.) Ordinates, percentages; abscissæ, ages.

ophthalmoscope (*vide* p. 110), or in high myopia large floating streamers. The normal muscæ volitantes are seen more plainly by myopic than by other eyes, probably because the entoptic image is generally larger.

A rare, but serious, change in the fundus is a small circular claret-coloured or black spot at the fovea. It may appear quite suddenly, being accompanied by great diminution of central visual acuity: it is probably due to intrachoroidal hæmorrhage or thrombosis.

Detachment of the retina is liable to occur spontaneously in about 5 per cent. of cases of high myopia, and not infrequently is bilateral (*vide* p. 378).

Ætiology. Myopia is rare in the new-born, the percentage of cases increasing rapidly during the first two decades, remaining constant afterwards at 10 to 15 per cent., as com-

pared with 30 per cent. for emmetropia and 50 to 55 per cent. for hypermetropia (Fig. 283). As regards sex, there is no doubt that the higher grades are commoner in women than in men. High myopia is as common among peasants as among the educated classes who do more near work. It is doubtful if near work is a cause of myopia, a view which has been held since the time of Kepler. There is, however, no doubt that it has a deleterious influence upon the disease. This has been attributed by Donders to (*a*) pressure of the extrinsic muscles upon the globe in strong convergence; (*b*) increased intraocular pressure from vascular congestion, due to the position of the head; (*c*) congestion of the fundus, leading to softening of the tissues. Accommodation has long been indicted as a cause of myopia. Many facts are against this view, *e.g.*, (*a*) accommodation occurs much more forcibly in hypermetropia; (*b*) it does not increase the intraocular pressure; (*c*) it does not affect the choroid farther back than the equator, and does not affect the sclerotic at all.

The mechanism whereby convergence influences the production of myopia is the subject of many theories, *e.g.*, pressure on the vortex veins, increased intraocular tension and so on.

Neither accommodation nor convergence alone suffices to explain the genesis of myopia. There must be some individual predisposition which provides any such accessory causes with advantageous conditions. There is probably a congenital weakness of the sclerotic. Other theories invoke shortness of the optic nerve (certainly false), special conformation of the skull leading to increased interpupillary distance or alteration in the position of the pulley of the superior oblique, &c. The view that moderate and high myopia are essentially distinct diseases, due to different causes, is probably untrue.

The cause of the myopic crescent has given rise to much discussion. It is probably congenital in origin, allied to other congenital crescents (*vide* p. 405), but there is no doubt that it may become altered by the conditions obtaining in the myopic eye. Anatomically there is considerable distortion of the papilla in myopia. It has been attributed to dragging, produced by the development of the posterior staphyloma; whether caused thus or not, it is an influential factor. Some authors ascribe the crescent to this cause (distraction crescent). In well-marked cases the head of the nerve is pulled over to the temporal side. The retina, including the pigment epithelium, is pulled slightly over the nasal edge of the disc (supertraction crescent). On the temporal side the pigment epithelium stops

short at a variable distance from the disc and the choroid is atrophic here (Fig. 285). This part appears ophthalmoscopically as the crescent.

The fact that the crescent may be absent in high myopia and is often present in low militates against the view that it is caused entirely by traction. It is not due to accommodation for the same reasons that myopia is not caused by this factor.

As regards *prognosis*, low or moderate degrees of myopia (up to 5 or 6 D), unless occurring in young children, have a good prognosis (*vide* p. 520). They are not likely to progress, and in some of the conditions of civilised life they may even be an advantage to the individual. The same condition in a child

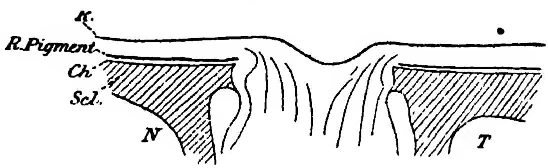


FIG. 284.

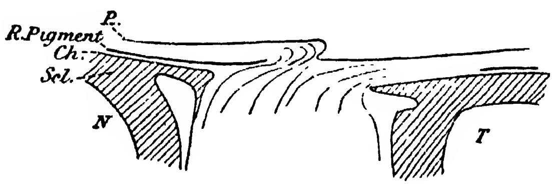


FIG. 285.

FIG. 284.—Diagrammatic horizontal section of normal disc.
FIG. 285.—Diagrammatic horizontal section of myopic disc. *N*, nasal side; *T*, temporal side; *R. Pigment*, retinal pigment epithelium; *R*, retina; *Ch.*, choroid; *Scl.*, sclerotic. (Modified from Heine.)

before the age of schooling, is of grave prognosis, because it is almost certain to progress, so that in a few years there may be 10 or 15 D of myopia, accompanied by serious fundus changes and defects of vision. The prognosis in high degrees of myopia is always grave. It must be judged by the acuity of vision after correction and the condition of the fundus. In all cases there is some danger of retinal detachment occurring.

Treatment consists in wearing suitable correcting glasses and attention to the hygiene of the eyes. Each case must be considered on its merits.

As regards the ordering of glasses in myopia, every surgeon agrees that *myopia must never be over-corrected.* Opinions differ as to details. In low myopia, up to 5 or 6 D (young

children excepted), no harm is done by ordering the full distant correction for constant use, but if this is done the patient must be warned not to hold near work closer than ordinary reading distance. Many surgeons order glasses weaker by 2 or 3 D for near work. This has the effect of making the patients artificially presbyopic, *i.e.*, if they hold the work at reading distance they exert 2 or 3 D less accommodation than an emmetropic person would do, or than they themselves would do if wearing full correction. Many patients are more comfortable for near work with the weaker glasses; others find no benefit. There is no doubt that the principle is derived from the fallacy that accommodation, *per se*, has a deleterious effect upon myopia. But there are some inherent objections to the weaker glasses. The patients often bring their work closer than reading distance. As far as accommodation is concerned this matters less with the weak glasses than with the strong, but mere accommodation is of little importance. It is convergence which is the important factor. In order to read at reading distance there must be some convergence. If the artificial presbyopic correction is given no stimulation to converge is supplied by the act of accommodation, so that in order that physiological requirements may be satisfied the visual axes should be parallel. This can only be effected by combining the glasses with prisms, bases in (*vide* p. 550). If the work is held too near still more convergence is required, and the arguments apply still more strongly.

In general, in low myopia, the full correction may be ordered for constant use, with minute instructions as to near work; in the event of any discomfort being experienced, weaker glasses should be ordered for near work, especially if much reading and sewing, &c., is engaged in. Children should wear their distance corrections, and wear them constantly—not specially in the interests of their eyes, but in the interests of their mental development. For children with even low degrees of uncorrected myopia cannot be expected to take a normal interest in their surroundings, since they cannot see distant objects as clearly as their fellows. Their mental horizon is constricted, they tend to become unduly introspective, and they are thrown more and more into finding their interest in reading and near work, so that it becomes more difficult than ever to restrict such work.

In young people with intermediate grades of myopia it is well to err on the safe side and order a slight under-correction.

In such cases, however, the patients will often peer obliquely through the glasses, which improves the definition in spite of the astigmatic effect of tilting strong lenses.

In high myopia it is wise always slightly to under-correct even for distance, and the same or still weaker glasses may be ordered for near work. In the highest grades the patient often sees best with glasses which are decidedly weaker than the full correction; he should be allowed to choose those he prefers. One reason is that the strong minus glasses very markedly diminish the size of the retinal images and make them very bright and clear. The retinal images are diminished because the glasses have to be worn farther from the eye than the anterior focal plane (*vide* p. 42); glasses for high myopia should therefore be made to fit as close to the eyes as possible: toric lenses may be ordered, or the eyelashes cut in order to prevent them from rubbing upon the glass. The very bright, clear images are uncomfortable because the retina is unduly irritable, probably owing to the fact that it has become accustomed to large indistinct diffusion images. Moreover, much artificial astigmatism, and therefore distortion of the image, is produced by looking obliquely through strong glasses; it is found to be most disconcerting to those who begin the use of glasses or have them much strengthened late in life. Very short-sighted people get into the habit of turning the head rather than the eyes to avoid looking obliquely through the glasses. Some high myopes can find their way about much better without any glasses. Contact glasses afford relief in cases where they can be borne.

In very high myopia the requisite amount of convergence for near work may be impossible. Reading and other near work then becomes purely uniocular. Generally one eye is better than the other, and this eye is always used. The effort to maintain convergence under impossible conditions is soon given up, which results eventually in the disused eye becoming divergent. There are other factors which tend to cause divergent strabismus in myopia (*vide* p. 584).

As regards hygienic measures in myopia, especially in the young, near work, apart from being held in the proper position, should be restricted. It is particularly important that it should not be done too continuously. More work can be done, with less harm to the eyes, by the interpolation of frequent short intervals of rest. It is best to give precise instructions as to the amount and distribution of near work. The illumination must be good, but not too bright, and it

should come from behind and beside the patient's head. If the light is bad there is a strong tendency to bring the book or work closer to the eyes in order to enlarge the retinal image. Reading in bed and stooping over near work must be forbidden.

The education of young children suffering from high myopia presents considerable difficulty. Most of the teaching is oral; the school books are printed in large characters, and writing is taught with bold letters on a blackboard. The methods adapted in the London County Council "myope classes" are admirable for the higher degrees, but are liable to be overdone by enthusiasts. Great judgment is needed in the restriction of near work in school children with 8 to 10 D of myopia. They are often unusually intelligent, and it must be realised that extreme measures, such as abstention from all near work for two or three years, may ruin their future careers.

If the eyes are irritable, or the myopia is progressing, complete rest is imperative. Atropine should be instilled once a day for a prolonged period, tonics, especially iron and arsenic, should be given, and a change of air to the country, with plenty of healthy exercise, is desirable. Excessive muscular exercise, straining, and lifting heavy weights should be avoided.

Operative treatment for high myopia. If an eye has axial myopia of 21 D, its length will be about 31 mm. (*vide* p. 533). If the crystalline lens of such an eye is removed, parallel rays will be focussed upon the retina without the intervention of any correcting lens, and the retinal images of distant objects will be larger than those of the emmetropic eye. Hence the extraction of the lens has been strongly advocated in high myopia. In completely successful cases the improvement is very great. The operation is, however, attended with grave dangers. The eye with high myopia is a diseased eye, which withstands operative measures badly. The vitreous is likely to be fluid and to contain opacities. The retina and choroid are probably diseased, and the tendency to detachment of the retina is increased by operation. No dogmatic rules can yet be given for the operation. I am guided by the following principles. (1) Only young patients should be operated upon; (2) the operation should be discission without subsequent curette evacuation unless it becomes imperative on account of tension; (3) there must be at least 15 D of myopia; (4) the fundus must be fairly healthy; (5) one eye only must be operated upon. The operation may be performed under less favourable circumstances if vision is so bad as to be useless, but such cases are rare.

Hypermetropia (*Syn.—Hyperopia*), or "far sight," is that dioptric condition of the eye in which, with the accommodation at rest, incident parallel rays come to a focus posterior to the light-sensitive layer of the retina. Hypermetropia may be due theoretically to any of the following conditions: —*A*. Abnormal shortness of the eye—*axial hypermetropia*. *B*. Abnormal curvature of the refracting surfaces—*curvature hypermetropia*: (*a*) too slight curvature of the cornea; (*b*) too slight curvature of one or both surfaces of the lens. *C*. Abnormal refractive index of the media—*index hypermetropia*: (*a*) too low index of the cornea or aqueous; (*b*) too low total index of the lens, due to (α) too low index of the nucleus, (β) too high index of the cortex, (γ) both these causes; (*c*) too high index of the vitreous. *D*. Abnormal position of the lens, *i.e.*, displacement backwards. *E*. Absence of the lens—*aphakia*. *F*. A combination of the above abnormalities.

As in myopia, the chief factor in clinical hypermetropia is abnormality in the length of the eye; in this case the eye is too short. It must be remembered that a small eye, though too short, is not necessarily hypermetropic, since there may be uniform diminution of all the parts. This is, perhaps, most easily understood if a diagram such as Fig. 29 is considered; if such a diagram is uniformly diminished, *e.g.*, by photography, the parallel rays will still come to a focus on the retina. As a matter of fact hypermetropic eyes are almost invariably also smaller than normal, a fact which is of great pathological importance (*vide* p. 285).

Curvature hypermetropia occurs commonly as a factor in astigmatism; it is almost unknown as a cause of spherical hypermetropia. Index hypermetropia accounts for the hypermetropia of old age (*vide* p. 53), and it is to be attributed to increased refractive index of the cortex of the lens.

Hypermetropia rarely exceeds 6—7 D, which is equivalent to a shortening of the optic axis of 2 mm. Individual cases of much higher degrees, without other anomaly, such as coloboma or microphthalmia, have been recorded—up to 24 D.

In the young the condition may cause no symptoms. When symptoms are present or arise, they are chiefly referable to the abnormal amount of accommodation to which these eyes are subjected, and to the lack of consonance between accommodation and convergence (*vide* p. 549). As has been pointed out, the healthy youth has an ample reserve of accommodation, and if he happens to be hypermetropic he accommodates for distant and near objects without being conscious of the act.

If he is weakly or does much near work the perpetual overaction of the ciliary muscle is likely to produce symptoms; the condition is often called accommodative asthenopia or "eye-strain." The symptoms are noticed chiefly after reading, sewing, &c., especially in the evening by artificial illumination. The eyes ache and burn; they may feel dry, so that blinking movements are more frequent than usual, or there may be lacrymation. The conjunctiva and edges of the lids become red, and actual blepharitis may be caused. If near work is persisted in, headache, usually frontal, comes on. Typical migraine may occur.

In young children hypermetropia is a predisposing cause of convergent strabismus (*q.v.*). In all cases latent convergence is often found in hypermetropes, though other forms of heterophoria may occur (*vide* p. 588). The presence of heterophoria increases the tendency to headache, &c.

In older patients no symptoms may be caused until the power of accommodation has diminished to the extent that the far point is beyond the range of comfortable reading distance. Near work has to be held farther off than usual in order to be seen clearly. The greater the degree of hypermetropia the sooner will this symptom arise. In other words apparent presbyopia commences at an earlier age than usual. It must be carefully borne in mind that hypermetropia predisposes to glaucoma (*q.v.*) in elderly people.

Ophthalmoscopically the fundus may exhibit no abnormality. A bright reflex, suggesting the appearance of watered silk, is commoner in hypermetropic than in emmetropic or myopic eyes. The inferior crescent is also more common in these eyes than in others, as also abnormal tortuosity of the retinal vessels. In some cases optic neuritis is nearly simulated—pseudo-papillitis (*vide* p. 390).

Anatomically the eye is shorter than normal in hypermetropia: it is also usually smaller. The changes are not confined to the post-equatorial segment as in myopia. The diameter of the cornea is often reduced, and regular astigmatism is common. The anterior chamber is shallower than normal, owing partly to the normal size of the lens (*vide* p. 285). Little weight should be attached to the old observation that the circular fibres of the ciliary muscle are hypertrophied, the meridional atrophied in hypermetropia. No anatomical abnormalities are found in the retina, choroid or optic nerve.

The new-born are almost invariably hypermetropic (mean 2·5 D). In the first decades of life the hypermetropia curve

falls rapidly, remaining at about 50 per cent. after the twentieth year (Fig. 283). Hypermetropia shows no predilection for either sex. It is a well-known fact that savages are usually hypermetropic. The higher mammals, especially the carnivora, are hypermetropic.

Treatment consists in prescribing the correcting glasses. Unless there are definite symptoms there is no reason for insisting upon the use of glasses in the young or middle-aged. In elderly people the hypermetropia must be corrected for near work: the ordinary presbyopic correction must be added to the hypermetropic correction, but care should be taken that these cases are rather under- than over-corrected (*vide* p. 539).

In young children the requisite correction is estimated under atropine, confirmed if possible by subjective tests. The correction, allowing for the effect of atropine upon the tone of the ciliary muscle (*vide* p. 516), is ordered for constant use or only for near work according to the severity of the symptoms. If the degree of hypermetropia is high the use of the glasses may be commenced while the child is still under the influence of atropine. In older patients with high hypermetropia it is often unwise to order the full correction at once. The ciliary muscle has been overworked so long that complete relaxation does not occur immediately. If the full correction is ordered the eye, with its contracted ciliary muscle, *plus* the glass, is made myopic: the patient cannot see clearly at a distance, and is liable to discard the spectacles. In these cases rather more than the amount of manifest hypermetropia should be ordered. The patient is told to return in three or six months, when stronger glasses are ordered, and so on until the full correction can be borne with comfort.

Astigmatism is that condition of refraction in which a point of light cannot be made to produce a punctate image upon the retina by any spherical correcting lens. The varieties of regular astigmatism have been already enumerated (*vide* p. 46).

Regular astigmatism, the only form which permits of optical correction, invariably produces greater or less defect in visual acuity. It is particularly liable to cause the worst forms of asthenopia or "eye-strain"; the asthenopia in these cases is only in part accommodative. It is often worse in the lower degrees of astigmatism than in the higher. This is probably due to the eye endeavouring so to accommodate as to produce a circle of least diffusion (*vide* p. 46) upon the retina. Aching of the eyes, severe headaches, and typical migraine are com-

plained of; the eyes quickly become fatigued with reading, and the letters are described as "running together."

Regular astigmatism is usually a congenital defect, due in most part to difference in curvature of the cornea in different meridians. It must be remembered that frequently the cornea is not alone at fault. Corneal astigmatism may be increased or partially corrected by lenticular astigmatism: hence the methods for correcting astigmatism, such as the ophthalmometer, &c., which are wholly dependent upon estimation of the corneal defect, are quite untrustworthy except in aphakia.

Regular astigmatism may be traumatic, following a wound, usually surgical, in the corneo-scleral margin. The contraction of the scar causes flattening of the cornea in the meridian at right angles to the wound. The astigmatism due to this cause continues to alter for many weeks after the injury, so that glasses should not be ordered for at least six weeks.

The higher degrees of astigmatism cause much lowering of visual acuity: this is usually least in mixed astigmatism, probably because the circle of least diffusion falls upon the retina.

Treatment. In all cases in which astigmatism causes asthenopic symptoms the full correction should be ordered for constant use, *i.e.*, both for distant and near vision. If there is a high degree of hypermetropia or myopia, associated with a low degree of astigmatism, the effect of the cylinder upon distant vision should be tested. If it produces no appreciable improvement simple spherical glasses should be tried first. It should be remembered that glasses placed before the eyes only correct the refraction accurately when the visual axis passes through the optical centre of the lens. When the eyes are directed to one side the lenses also act as prisms, and further the lenses are tilted relatively to the eyes so that an astigmatic effect is produced. In the case of high spherical lenses the astigmatic effect is considerable, and may easily counteract or double the effect produced by a weak cylinder combined with the sphere. Hence weak cylinders are seldom of much use when combined with high spheres.

In low astigmatic errors the instruction as to the use of glasses depends upon the amount of asthenopia. The relief of the discomfort experienced may not be worth the trouble of wearing glasses constantly. In these cases they should be ordered for near work only, and if this fails to eliminate the symptoms the advice should be given to wear them constantly.

Aphakia is the condition of the eye when the crystalline lens has been removed. The eye is extremely hypermetropic if it was emmetropic or had only a low grade of ametropia before removal of the lens. The hypermetropia, as estimated by the correcting lens required when worn in the usual position, is about 10 or 11 D if the eye was previously emmetropic.

The optical conditions of the aphakic eye are very simple. It consists of a curved surface, the cornea, separating two media, air and vitreous, of different refractive indices. Knowing the radius of curvature (8 mm.) and the refractive indices (1 and 1·33), it is easy to calculate the focal distances. The anterior focal distance is 23 mm. and the posterior 31 mm., as compared with 15 mm. and 24 mm. respectively for the normal eye. If the aphakic eye were 31 mm. long parallel rays falling on the cornea would be brought to a focus on the retina and no correcting glass would be required for distance. It is easy to calculate the amount of axial myopia of a phakic eye which is 31 mm. long. It has been already pointed out (p. 121) that in the phakic eye 1 mm. of elongation is equivalent to an axial myopia of 3 D. Therefore an elongation of 31 — 24 mm., *i.e.*, 7 mm., equals 21 D (*vide* p. 528).

The retinal image of the aphakic eye is about a quarter larger than the emmetropic retinal image. Hence vision of 6/6 with a correcting glass after extraction is not quite so good as it seems.

Accommodation is, of course, lost. The anterior chamber is deep, the iris tremulous, and there is often a coloboma of the iris upwards. In cases of doubt as to the absence of the lens the Purkinje-Sanson reflexes from the lens surfaces should be sought.

With the ophthalmoscope opacities will probably be found in the pupillary area, consisting chiefly of remnants of the lens capsule. They should be examined by oblique illumination, by the mirror, and by the direct method. If they are dense, discission is indicated before attempting to correct the refraction; if they are slight, the advisability of needling depends upon the amount of vision obtained with correction.

In addition to the hypermetropia, there is always some astigmatism in those cases in which a corneal or corneo-scleral section has been made. If the section is in the upper part of the cornea, the astigmatism is against the rule; *i.e.*, the cornea is flattened in the vertical meridian. The astigmatism usually amounts to 2 or 3 D. It gradually diminishes, fairly quickly at

first and very slowly after the first few weeks, as the cicatricial tissue in the scar contracts.

Treatment. The refractive error is determined by retinoscopy and by subjective tests. The ophthalmometer may afford help in these cases. Great patience is often necessary, for the patients do not readily accommodate themselves to the new optical conditions. A 10 or 11 D convex lens combined with a + 2 or + 3 D cylinder, axis horizontal, is about the correction usually required for distance. It should be remembered that the lens in the trial frame is usually farther from the eye than in well-fitting spectacles. With these strong lenses an appreciable error is introduced, and the spheres ordered should be 0·5—0·75 D stronger than those which give the optimum result with the trial frame. The sphere must be stronger by 4 D for near work. A small amount of false accommodation can be obtained by slightly altering the distance of the glass from the eye. The correcting glasses should not be ordered earlier than six weeks after the operation, both on account of the necessity of resting the eye and because the astigmatism changes rapidly during the first few weeks.

If one eye only has been operated upon, the other being cataractous, reversible spectacle frames may be ordered. In them the bridge is horizontal, so that when the distant glass is being used the near glass is in front of the eye which has not been operated upon, and *vice versâ*. Reversible frames, however, never fit very satisfactorily.

The aphakic eye is specially liable to erythropsia (*q.v.*) and should therefore not be exposed to very bright light.

Anisometropia is the condition in which the refraction of the two eyes shows a considerable difference. A slight difference is very common. The condition may cause asthenopic symptoms. All varieties and degrees of anisometropia occur. In the lower grades there is usually binocular vision, though it is imperfect. In the higher grades this is impossible without correction. Distinct vision is then uniocular, and there is some danger of the eye which is not used becoming divergent. If one eye is nearly emmetropic and the other myopic, the former may in some cases be used for distant, the latter for near, vision.

Treatment. Correction of anisometropia offers many difficulties. It has already been mentioned that if correcting glasses are placed at the anterior focal plane of the eye, the retinal images in axial ametropia are the same size as the emmetropic retinal image. In practice the glasses are farther

from the eyes. Consequently with convex glasses the retinal image is enlarged, with concave diminished. In high grades of anisometropia, therefore, there will be a considerable difference in the size of the retinal images of the two eyes (*aniseikonia*). Patients find it difficult or impossible to fuse these sharp but diverse images. Moreover, on looking obliquely through the glasses the prismatic effect and the distortion are different in the two eyes, enhancing the discomfort. Contact glasses diminish these optical effects, and may be ordered in suitable cases.

No universal rules can be given for the glasses which should be ordered. The following suggestions will generally be found to work well: If the difference between the two eyes is less than 4 D, the full correction should be ordered for constant use; they should be perseveringly worn for at least six weeks. If still they cannot be borne, it will probably be necessary to correct only the less ametropic eye for distance.

In patients of less than twelve years of age the full correction should also be ordered for constant use, even if the difference is greater than 4 D. The more ametropic eye should be exercised alone, as in cases of concomitant strabismus (*vide* p. 580). Very often the treatment will fail, but it should be tried in the interests of binocular vision. It is almost certain to fail in older patients.

When the full correction cannot be worn constantly and one eye is myopic, both eyes can often be made to work together in comfort for near work by making each eye artificially presbyopic to the extent of half the difference between the two eyes. For example, suppose one eye is emmetropic and the other has 3 D of myopia, the patient will be most comfortable with + 1·5 D in front of the emmetropic, and − 1·5 D in front of the myopic eye for near work.

The Correction of Errors of Refraction.

The correction of errors of refraction has been already briefly sketched. It will be well, however, to outline the method to be adopted in systematically examining for and correcting these errors, and to indicate the requirements which should be satisfied by spectacles.

If the patient is less than fifteen years of age,—

(1) Test the distant and near vision, if the child knows his letters;

(2) Test the pupil reactions;

(3) Test the muscular balance by the screen test (*vide* p. 553);

(4) Examine the fundi with the ophthalmoscope.

Then order ung. atropinæ, 1 per cent., to be inserted with a glass rod, three times a day for at least three days.

At the next visit—

(1) Determine the error of refraction by retinoscopy;

(2) Thoroughly examine the fundus with the ophthalmoscope;

(3) Confirm the retinoscopy by subjective tests, if the child knows his letters;

(4) Order the correction according to the principles enunciated in the paragraphs devoted to the respective types of refractive error.

If the patient is between fifteen and twenty-five years of age, the same procedure should be adopted, but in many cases the prolonged action of atropine so seriously interferes with the patient's employment that it may be replaced by homatropine.

If the patient is between twenty-five and forty,—

(1) Test the distant vision, the manifest hypermetropia, and the near vision;

(2) Test the pupil reactions and the range of accommodation;

(3) Thoroughly examine the eyes with oblique illumination and by the ophthalmoscope.

If it is concluded that the defect is simply due to error of refraction, the further procedure depends upon the results of the subjective testing :—

(1) If the vision is 6/6 and J. 1, with a low degree of manifest hypermetropia and few asthenopic symptoms, glasses may be ordered according to the amount of manifest hypermetropia (*vide* p. 531).

(2) If the vision is less than 6/6 no Hm, but J. 1 is read fluently when the type is held closer to the eyes than normal, the patient has probably simple myopia. In general homatropine should be instilled, and the glasses ordered according to the retinoscopy and subjective tests under the mydriatic. The expert may feel justified in ordering glasses without using a mydriatic, judging by the subjective tests and his ophthalmoscopic examination, confirmed by retinoscopy without a mydriatic.

(3) If the vision is less than 6/6, and 6/6 cannot be read with any spherical glass, or if some letters only of 6/6 can be read—letters with oblique lines, *e.g.*, Z, being missed in that line and even in the other lines—the patient is probably astigmatic.

Homatropine should be instilled, and the refraction corrected by retinoscopy.

If the patient is over forty, the examination will be exactly as for one between twenty-five and forty, except that presbyopia must be taken into account, and greater care must be exercised in instilling a mydriatic. Presbyopia affects the distant vision in hypermetropes in such a manner that although 6/6 may not be read with the unaided eye, it may be possible with a convex lens. Its effect upon near vision is discussed elsewhere. It is of little use to test the near vision of a presbyope without the glass which is necessary to correct the presbyopia, since no useful knowledge is obtained. If the vision cannot be improved up to 6/6 with a spherical lens, the patient is probably astigmatic, or has some disease of the eye, *e.g.*, incipient cataract. If ophthalmoscopic examination indicates merely the presence of astigmatism, homatropine (*never atropine*) should be instilled, but *in every case in which this is done one drop of eserine,* 0·5 *per cent., is instilled into the eye before the patient leaves.* After estimating the error of refraction by retinoscopy it is advisable to see the patient again after the effects of homatropine have passed off before ordering glasses for near vision.

Spectacles. In children spectacles with large round or " round oval " " eyes " should be ordered, otherwise the child may look over them.

In adults with astigmatism spectacles or rigid pince-nez must be ordered, never " folders." The latter are never to be recommended, and are absolutely contraindicated in astigmatism.

It is very important that all glasses fit accurately. In distant glasses the lenses must be centred so that the optical centres are exactly opposite the centres of the pupils when the visual axes are parallel. Near glasses are decentred slightly inwards, and the lenses are tilted so that the surfaces form an angle of 15° with the plane of the face: they are then approximately at right angles to the visual axes when the eyes are directed downwards in reading.

Various forms of bifocal glasses are sometimes used. In them the upper part contains the distant correction, the lower part the near. If recommended, patients should be warned that they may experience some difficulty in going downstairs and so on, since objects on the ground will appear blurred when looking through the glasses and prismatic effects cause apparent displacement of objects.

If tinted glasses are desirable, *e.g.*, in high myopia, albinism, &c., the correcting lenses may be tinted. For use in tropical countries Crookes's glass (*vide* p. 148) may be used.

In cases of irregular corneal astigmatism and high myopia great improvement of vision occurs when a suitably curved glass meniscus is placed in actual apposition to the cornea. Such "contact" glasses cannot be borne in all cases and much perseverance may be required in their application and removal. They are not free from danger of causing ulceration,

CHAPTER XXV

Anomalies of Accommodation

Presbyopia has been already explained (*vide* p. 55). It is a physiological condition and therefore not properly included amongst anomalies of accommodation. It is convenient, however, to discuss its clinical treatment here, since it is so nearly allied to the correction of errors of refraction.

It has been shown that no convex lens of greater strength than 4 D should be ordered to correct presbyopia in the absence of hypermetropia; further, that the rule that a presbyope requires + 1 D for every five years after forty errs on the side of being a somewhat liberal allowance. Rather less, and never more, should be ordered. Some people, especially if they have hypermetropia, and therefore still stronger glasses for near work, have discomfort with their proper presbyopic correction. It is usually due to the absence of any stimulus, derived from the accommodative effort, to converge (*vide* p. 549). Theoretically the visual axes should be parallel when the presbyopic correction is used. Convergence, however, is necessary in order that both eyes may see the near object. It may be eliminated by combining prisms, bases in, with the correcting glasses.

In many occupations, *e.g.*, bootmaking, carpentering, and so on, the work is held at a greater distance than ordinary reading distance. The correction for work must then be ordered according to the distance, a weaker glass being required for a distance greater than 22 cm.

Myopes of course may require no glass for near work. Their presbyopic correction is estimated by the algebraic sum of their myopia and presbyopia.

Paralysis of Accommodation, or *cycloplegia*, occurs in disease as well as from the direct action of drugs (cycloplegics) such as atropine and homatropine. Unilateral cycloplegia is generally due to drugs (often through rubbing the eyes after using a belladonna liniment), contusion (*vide* p. 441), or to paralysis of the third nerve. Bilateral paresis, less commonly paralysis, is most frequent after diphtheria, but may occur after debilitating

illness, influenza, syphilis, diabetes, tabes, cerebral disease, &c. Paresis of accommodation occurs as a premonitory symptom of glaucoma.

In complete paralysis the sphincter pupillæ is also generally paralysed, so that the pupil is widely dilated. In paresis the pupil may be scarcely at all affected, especially after diphtheria, but in this disease the reverse of the Argyll Robertson pupil may be met with, viz., loss of reaction to accommodation with retained reaction to light. The symptoms depend upon the condition of the refraction. If the patient is myopic, the defect may pass quite unnoticed; if he is emmetropic, near vision will be alone affected; if he is hypermetropic, both distant and near vision will be affected, but particularly the latter. In paresis it may be possible to diagnose the condition only by carefully measuring the range of accommodation.

In diphtheritic cases the paralysis of accommodation follows the primary attack at an interval of several weeks, and is often associated with paralysis of the palate, loss of knee jerks, &c. The sore throat may have been very slight and its diphtheritic character unrecognised. The lesion is probably nuclear, either toxic or hæmorrhagic. Cycloplegia in middle life should arouse suspicion of diabetes. It also occurs in chronic alcoholism.

Paralysis of accommodation in children is liable to be overlooked owing to instillation of atropine for estimating errors of refraction without previously testing the near vision.

The prognosis is good in cases due to drugs or diphtheria. In traumatic cases the condition may be permanent.

Treatment is that of the cause. Post-diphtheritic cases should be treated with tonics, especially strychnine. Whenever the condition is bilateral near work can be carried on by using suitable convex glasses, as in the correction of presbyopia. As a rule, however, the eyes should be kept at rest, so that it is inadvisable to order glasses. Miotics are sometimes used, but they may do harm and seldom do good. The constant current may be tried.

Spasm of Accommodation. It has already been mentioned that the ciliary muscle has physiological tone, which is abrogated by atropine, and is equivalent to about one dioptre. In some cases it is found that atropine produces a much greater effect. This can only be due to spasm of the ciliary muscle. It is found only in young patients, and, contrary to what might be expected, more often in myopes than in hypermetropes. In any case an actual or relative myopia is produced.

Spasm of accommodation is produced artificially by the instillation of miotics.

In spontaneous spasm of accommodation there is nearly always some error of refraction. The eyes have usually been subjected to too much near work under unfavourable circumstances. The condition should not be diagnosed unless proved to be present by the use of atropine.

Treatment consists in the use of atropine for several weeks. The amount of near work must be limited and carried out under good conditions, the error of refraction being carefully corrected.

SECTION V

DISORDERS OF MOTILITY OF THE EYE

CHAPTER XXVI

Anatomy and Physiology of the Extrinsic Ocular Muscles

THE internal rectus is inserted into the sclerotic about 5·5 mm. to the nasal side of the corneo-scleral margin, the inferior rectus 6·5 mm. below, the external rectus 7 mm. to the temporal side, and the superior rectus 7·5 mm. above (Fig. 286).

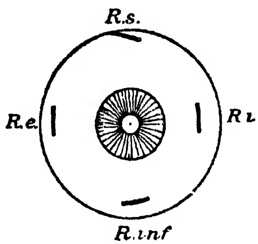


FIG. 286.—Lines of insertion of the recti muscles of right eye seen from in front.

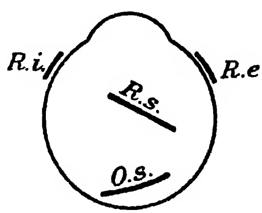


FIG. 287.—Lines of insertion of the superior oblique muscle and of the superior, external and internal recti of right eye, seen from above.

The tendons are about 10 mm. broad. The origin of these muscles around the optic foramen is much to the nasal side of the posterior pole of the eye. It has been proved that when the extrinsic muscles act they turn the eye around a spot which is called the centre of rotation (Fig. 288). This spot is situated about 13·5 mm. behind the centre of the cornea. It lies in the same horizontal plane as the lateral recti. Con-

sequently when the internal or external rectus acts it rotates the eye horizontally inwards or outwards respectively around a vertical axis through the centre of rotation without any rotation about the horizontal axis. When, however, the superior rectus acts, it not only pulls the eye upwards, but also inwards, while there is some rotation of the cornea, so that the vertical meridian assumes a direction from above down and out (torsion). Similarly when the inferior rectus acts the eye is pulled down and in, the vertical meridian of the cornea being deviated so that it lies from above down and in (Fig. 289).

The oblique muscles are inserted into the sclerotic behind the level of the centre of rotation (Fig. 287). Their direction of action is from behind forwards and inwards. Hence the superior oblique pulls the eye downwards and outwards, the inferior oblique upwards and outwards. The mechanism is so arranged that when the superior rectus and inferior oblique act simultaneously the eye moves directly upwards; *i.e.*, the upward movement caused by each muscle is summated, while the inward movement and corneal rotation of the superior rectus are exactly compensated by the outward movement and contrary corneal torsion of the inferior oblique. Similarly when the inferior rectus and superior oblique act simultaneously the eye moves directly downwards.

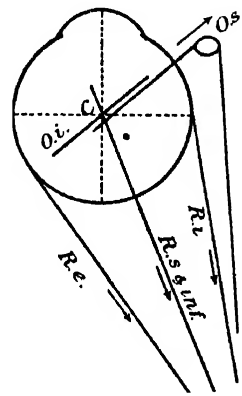


FIG. 288.—Diagram of the lines of action of the extrinsic muscles of left eye. (After Fick.) *C*, centre of rotation. The arrows show the directions of action of the muscles. *R.e.*, external rectus; *R.i.*, internal rectus; *R.s. & inf.*, superior and inferior recti; *O.s.*, superior oblique; *O.i.*, inferior oblique.

Every movement of the eyeball is a synkinesis (*vide* p. 565). In adduction not only does the internal rectus act, but also the superior and inferior recti, and it has been shown that the antagonistic muscles are not merely relaxed, but are actively inhibited. In abduction the external rectus and both obliques are in action. In elevation the superior rectus acts consonantly with the inferior oblique. In depression the inferior rectus acts with the superior oblique. The movements already described are all around three primary axes—vertical (move-

ments in and out), coronal (movements up and down), and sagittal (torsion)—which pass through the centre of rotation. Still more complicated are the movements about secondary axes, *i.e.*, axes passing through the centre of rotation in some other direction, such as movements up and in, up and out, down and in, down and out.

Not only is there uniocular synkinesis: under normal circumstances there is always also binocular synkinesis. Abduction of one eye is accompanied by adduction of the other eye—conjugate deviation; elevation or depression of one eye is always accompanied by elevation or depression respectively of the other eye. The only exception to this rule is the bilateral

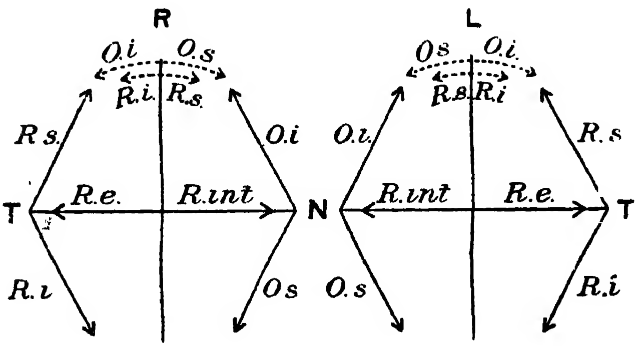


FIG. 289.—Diagram of the lines of action of the extrinsic muscles upon the corneæ. (Elschnig.) R, right eye; L, left eye; T, temporal sides; N, nasal sides. The dotted lines show the torsional effects.

adduction of the eyes in convergence. Elevation of both eyes is accompanied by slight abduction (divergence), depression by slight adduction (convergence).

The oculomotor, or third cranial nerve, supplies all the extrinsic muscles except the external rectus and superior oblique; it also supplies the sphincter iridis and ciliary muscle. The superior oblique is supplied by the fourth nerve, and the external rectus by the sixth nerve. A thorough knowledge of the arrangement of the nuclei of the cranial nerves in the mid-brain and medulla, and of the course and relations of the nerves to their destinations, is requisite for accurate diagnosis of the seat of the lesion in cases in which they are involved.

The third and fourth nuclei form a large continuous mass of